STUDY ON DETERMINANTS AND CURRENT STATUS OF IMMINENT ECLAMPSIA AND ECLAMPSIA

DISSERTATION SUBMITTED FOR

M.S. (BRANCH II)

OBSTETRICS & GYNAECOLOGY

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

CERTIFICATE

This is to certify that this dissertation titled **"STUDY ON DETERMINANTS AND CURRENT STATUS OF IMMINENT ECLAMPSIA AND ECLAMPSIA"** submitted by **DR. MEENA. K** to the faculty of Obstetrics and Gynecology, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S. degree Branch II Obstetrics and Gynecology, is a bonafide research work carried out by her under our direct supervision and guidance from July 2013 to August 2014.

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DECLARATION

I, **DR. MEENA.K** solemnly declare that the dissertation titled **"STUDY ON DETERMINANTS AND CURRENT STATUS OF IMMINENT ECLAMPSIA AND ECLAMPSIA"** has been prepared by me. This is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the requirement for the award of M.S. degree (Branch II) Obstetrics and Gynecology. I also declare that this bonafide work has not been submitted in part or full by me or any others for any award, degree or diploma to any other university or board either in India or abroad.

Place: **DR. MEENA.K**

Date:

ACKNOWLEDGEMENT

I owe my thanks to **The Dean Dr.R.M.RAJAMUTHAIYAH M.S., MCH.,** Govt. Theni Medical College for permitting me to carry out this study at Theni Medical College.

I am deeply indebted to **Dr.M.THANGAMANI, M.D.,O.G..,** professor and Head of the Department of Obstetrics and Gynecology, Govt. Theni Medical College, Theni, for suggesting the topic, for her able guidance, inspiration and the encouragement she rendered at every stage of this study.

I am very grateful to Assistant Professor **Dr.A.MAHALAKSHMI, M.D., OG.,** for her valuable guidance in conducting and completing the study.

I express my gratitude to all **Assistant Professors Dr.B.Shanthi Rani, M.D.,OG., Dr.A.Shanthavibala, M.D.,OG., Dr.K.Kameshwari, M.D.,OG., Dr.J.Rekha, M.D.,OG., Dr.K.Moogambigai, M.D.,OG.,** Department of Obstetrics and Gynecology for their kind co-operation in helping me to do this study.

Last but not the least, I gratefully acknowledge my thanks to all my Colleagues and the co-operation of the patients without whom this study would not have been possible.

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INTRODUCTION

STUDY ON DETERMINANTS AND CURRENT STATUS OF IMMINENT ECLAMPSIA AND ECLAMPSIA

INTRODUCTION:

Eclampsia is a Greek word meaning “shining forth”. It is an acute obstetric emergency associated with both maternal and perinatal morbidity and mortality. Eclampsia is preventable but it is still one of the common causes of maternal mortality.

Incidence of Eclampsia in developed country is 1 in 2000 to 1 in 3448. Incidence of Eclampsia in India is 0.9 to 1.8%. The maternal mortality ratio in 2009 for India is 212 per 1 lakh with hypertensive disorder causing 5% maternal death. Perinatal Mortality has been as high as 59/1000 to 214/1000 and morbidity as high as 56%. About 35% of affected women have serious complication. Major maternal complication includes placental abruption – 10%, Neurological deficit – 7%, pulmonary edema – 5%, cardiopulmonary arrest – 4% acute renal failure – 4% and 1 % maternal death. The most common cause of fetal death are prematurity, fetal asphyxia and acidosis.

Severe pre-eclampsia with prodromal symptoms is called Imminent eclampsia.The prodromal symptoms are headache, epigastric pain, nausea, vomiting and blurring of vision. Eclampsia is the occurence of seizure or coma in pre-eclamptic women. Eclampsia can occur at any period of

pregnancy antepartum, intrapartum and postpartum. Antepartum eclampsia is more common.The classical symptoms of pre-eclampsia is hypertension and proteinuria. some cases may present without this classical symptoms called atypical pre-eclampsia/ eclampsia.

There are many determining factors affecting the maternal and perinatal outcome in eclampsia.Eclampsia is more common in antenatal mother who didn't have proper antenatal checkup. Eclampsia is common in low socio-economic group and various epidemiological factors affect the maternal and perinatal outcome. Eclampsia is common in primigravida and occurs more commonly in last trimester. Maternal and perinatal outcome also depend on nature of fits and how soon patient receive treatment and quality of treatment. Eclampsia is usually preceded by imminent symptom. By proper antenatal care, early detection of pre-eclampsia and by prompt management, eclampsia and its complication can be reduced.

AIM OF THE STUDY

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- **To analyse the determinants of Imminent eclampsia and Eclampsia**

- **To analyse the maternal and perinatal outcome in women with Imminent eclampsia and Eclampsia.**

- **To discuss the measures to prevent Eclampsia and its complication.**

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Eclampsia:

Eclampsia is defined as the development of seizure and or unexplained coma during pregnancy, labour or postpartum in patients with signs and symptom of pre-eclampsia.

Measurement of Blood Pressure:

- Blood Pressure is measured with the patient in sitting position and it is preferable to measure the blood pressure in the right upper arm. The arm is kept at level of heart.
- The cuff should be of appropriate size, the length should be 80% and width should be atleast 40% the arm circumference.
- The cuff size used are
    - Small adult : 22 – 26 cm
    - Adult : 27 – 34 cm
    - Large adult : 35 – 44 cm
    - Adult thigh : 45 – 52 cm
- The BP cuff bladder is placed in the arm in midline over the brachial artery pulsation and lower end 2-3cm above the antecubital fossa.
- The cuff is inflated above systolic pressure (disappearance of the radial pulse) and deflated slowly. The first sound heard by auscultation

(Korotkoff Phase I) is recorded as systolic BP and disappreance of sound (Korotkoff Phase V) is recorded as diastolic BP.

Classification of hypertensive disorder complicating pregnancy:

Classification according to working groups of the National High Blood Pressure Education Programme (2000)[1].

- Gestational hypertension
- Pre-eclampsia and eclampsia
- Pre-eclampsia superimposed on chronic hypertension.
- Chronic hypertension

Gestational hypertension:

- When the systolic BP $\geq$140mmHg and /or diastolic BP $\geq$90mmHg, measured on two occasion atleast 6hrs apart.
- Without Proteinura.
- Diagnosed after 20 weeks of gestation and BP should return to normal before 12weeks postpartum.

Pre-eclampsia:

Mild pre-eclampsia

- If systolic BP $\geq$ 140mmHg and diastolic BP $\geq$ 90mmHg after 20 wks of gestation.
- Protenuria of +1 dipstick or $\geq$ 300mg/24hrs

Severe pre-eclampsia

- If systolic BP $\geq$ 160mmHg and diastolic BP $\geq$ 110mmHg on two occasions 6hrs apart.
- If Proteinuria $\geq$ 5gm in 24 hrs urine sample or $\geq$3+ on two random urine sample.
- Urine output <500ml in 24 hrs
- Headache and visual disturbance
- Pulmonary edema or cyanosis.
- Epigastric or right upper quadrant pain.
- Impaired liver function.
- Fetal growth retardation.
- Thrombocytopenia.

Eclampsia

Eclampsia is the occurrence of seizure or coma in a pre-eclamptic women.

Superimposed pre-eclampsia on chronic hypertension

New onset proteinuria in hypertensive women who doesnot have proteinuria before 20 weeks.

A sudden increase in blood pressure or proteinuria in women with hypertension and proteinuria before 20 weeks of gestation.

Chronic hypertension

When blood pressure is >140/90mmHg before pregnancy or diagnosed less than 20 weeks of gestation.

Hypertension diagnosed after 20 weeks of pregnancy but persist even after 12 weeks postpartum.

Pre-eclampsia:

Two Stage Disorder:

Redman[2] and Colleague (2009) described pre-eclampsia as a two stage disorder. Stage 1 is preclinical caused due to faulty trophoblastic vascular remodeling of uterine arteries causing placenta hypoxia. Stage 2 is caused by release of placental soluble factors into maternal circulation causing systemic inflammatory response and endothelial activation.

Etiology:

Etiology of pre-eclampsia is culmination of factors involving maternal, fetal and placental factors.

- Abnormal trophoblastic invasion
- Immunological factor
- Maternal maladaptation to inflammatory signs of pregnancy
- Genetic factor

Abnormal Trophoblastic invasion:

Fisher[3] and colleagues, 2009: Normally by invasion of endovascular trophoblast, spiral arteries undergo extensive remodeling and vessel diameter increases. In pre-eclampsia due to incomplete trophoblastic invasion only deicidual vessel invaded and not the myometrial vessel. Hence deeper myometrial vessel do not loose their endothelial lining and musculo-elastic tissue and their mean external diameter is only half that of vessels in normal placenta.

Redman[4] and Sargent (2008): Placenta blood flow is impaired due to abnormally narrow spiral arteriolar lumen leading to hypoxic environment. Placental debris are released due to hypoxic environment leading to systemic inflammatory response and endothelial activation which leads to syndrome of pre-eclampsia.

Madazli[5] and Anociates (2000): Severity of hypertension correlates with the magnitude of defective trophoblastic invasion.

Immunological factors:

In normal pregnancy there is maternal immune tolerance to paternally deveried placenta and fetal antigen.

American journal[6] of obstetrics and gynaecology: (Vol. 180, issue 2 Feb.1999, Page 499 - 506) Pre-eclampsia is due to immunological maladaptive tolerance between maternal, paternal and fetal tissue.

Labarrere[7] (1988) studied the histology of placenta in pre-eclampsia and showed acute graft rejection at maternal placental interface.

Redman[2] and Collegues (2009) studied the role of immune maladaptation in pathophysiology of preeclampsia and showed that in pre-eclampsia the amount of immunosuppressive human leucocyte antigen G (HLA-G) expressed by extravillous trophoblast during earlier weeks of pregnancy was reduced. This leads to defective placental vascularization which contributed to stage I of pre-eclampsia. In early second trimester TH1 action was increased and TH1/TH2 ratio changes.Th1 cells stimulate inflammatory cytokine secretion. This immunological mediated inflammatory reaction were stimulated by placental debris and adipocytes. This inflammatory cytokines causes endothelial cell injury leading to pre-eclampsia.

Endothelial cell activation:

Fass[8] 2000: Activated leucocytes in maternal circulation cause endothelial cell dysfunction.

Manten[9] and associates 2005: Cytokines such as TNF-alpha and IL1 causes oxidative stress and release of free radicals that lead to formation of self propagation lipid peroxides. Lipid peroxides generate highly toxic radicals that injure endothelial cells, decreases nitric oxide production and alter the prostaglandins levels. Oxidative stress also leads to production of lipid laden macrophages, foam cells seen in artherosis. Activation of microvascular

coagulation causes thrombocytopenia. Increased capillary permeability causes edema and proteinuria.

Nutritional factor:

- **John[10] and coworker 2002:** Diet having antioxidant is associated with decreased blood pressure.
- **Zhang[11] and Associates (2002):** Dietary intake of ascorbic acid <85mg/day has double the risk to develop pre-eclampsia.
- **Villar[12] and Associates (2006):** Calcium supplementation to low dietary calcium intake population has no effect on incidence of pre-eclampsia.

Genetic factors:

Ward[13] and Lindheimer (2009): Incident risk of pre-eclampsia for daughter of pre-eclamptic women is 20 – 40% and sister is 11 – 37% and for twins are 22 - 47 %.

More than 70 genes have studied for their association with pre-eclampsia.

Seven of these are widely investigated. They are:

Chromosome	Gene	Biological association
1p 36.3	MTHFR (C677T)	Vascular disease
1q 23	F5(Leiden)	Thrombophilia
1q42, q43	AGT (M235T)	Blood pressure regulation
6q 21.3	HLA (Various)	Immunity
7q 36	NO53 (alu298 Asp)	Vascular endothelial function
11p 11q 12	F2(G20210A)	Prothrombin (factor 11)
17q23	ACE (1/ D^{at} intron 16)	Blood pressure regulation

Pathogenesis:

Vasospasm:

- The concept of vasospasm was studied as early as 1918 by Volhard.
- Vascular constriction leads to increase vascular resistance and subsequently hypertension. Additionally endothelial damage cause interstitial leakage through which blood constituents, including fibrinogen and platelets are deposited subendothelially. The effect of diminished blood flow causes ischemia of surrounding tissue which leads to necrosis, hemorrhage and end organ damage (characteristic of syndrome).

Endothelial cell activation:

- **Grundmann[14] and Associates (2008):** Reported that circulating endothelial cells are fourfold elevated in pre-eclamptic women.

Increased pressor response:

- **Raab[15] and Co worker 1956: Talledo and associates 1988:**Women with Pre-eclampsia have increased vascular response to infused angiotenin II and Norepinephrine.
- Increased sensitivity to angiotensin II clearly precedes the onset of gestational hypertension.

Role of Prostaglandin:

Spitz[16] and Collegues, 1988: Thromboxane A2 secretion in pre-eclampsia is increased and Prostacyclin: Thromboxane A2 ratio decreases resulting in increased sensivity to infused angiotensin II and ultimately vasoconstriction.

Nitric oxide:

Decreased endothelial nitric oxide synthase expression cause decreased nitric oxide production leading to increased mean arterial pressure.

Endothelin:

Pre-eclampsia women have higher level of endothelin – I **(Ajne[17]-2003).** Endothelin is potent vasoconstrictor.

Angiogenic and antiangiogenic factor:

- SFLT – 1- Soluble Fms Like Tyrosine kinase -1 is a variant of Flt – 1 which is the receptor for vascular endothelial growth factor (VEGF) and placental growth factor. SFLT – 1 level increases in maternal serum of pre-eclampsia women before several months.
- **Maynaud[18] and Associates 2003:** Increased SFLT – 1 inactivate and decrease circulating free PLGF and VEGF leading to endothelial dysfunction.
- **Levine[19] and Co-worker 2006:** Soluble endoglin (SEng) also called CD105 is derived from placenta blocks endoglin – coreceptor for TGF-β family. This inhibits various TGF-β isotopes from binding to endothelial receptors. Subsequently decreased endothelial nitric oxide dependent vasodilatation.

PATHOPHYSIOLOGY:

Placenta:

- Placenta shows infarcts , congested chorionic villi, hematomas, proliferative endarteritis and degeneration.
- Microscopic examination shows increased syncitial knots, endothelial proliferation, cytotrophoblast, cellular proliferation, fibrinoid necrosis, calicified and hyalinised villous spot.

Kidney:

- In preeclampsia there is glomerular and tubular dysfunction. Characteristic feature is glomerular endotheliosis, in which glomeruli are diffusely enlarged and avascular. Microscopy reveals exudation of foamy macrophages, lymphocytes, polymorphs and nuclear leucocytes in the capillary lumen and mesangium. This narrow the lumen and causes decreased glomerular filtration rate. Acute tubular necrosis is rare and it may be reversible. Rarely acute cortical necrosis may occur which is irreversible.

Liver:

- The pathological change in liver is periportal thrombosis, fibrin deposition, haemorrhage and necrosis. Small haemorrhage may coalesce to form subcapsular hematoma causing stretching of Glisson capsule and rarely causes liver rupture.

Cardiovascular changes:

- Increased cardiac afterload due to hypertension
- Hypervolemia of pregnancy is dimnished in preeclampsia causing decreased cardiac preload.

- Endothelial cell activation causes increased capillary permeability leading intravascular fluid to enter in to extravascular space. This may cause pulmonary edema despite normal ventricular function.

Brain:

The main finding in the brain is cerebral vasospasm. Secondary to endothelial dysfunction small cerebral hemorrhages, thrombosis and fibrinoid necrosis can occur. Cerebral edema is usual in eclampsia. when there is widespread edema patient comatose.

Eyes:

- Localised retinal vasospasm is the most common finding. Haemorrhage and papilledema are seen in severe hypertension. Visual disturbance are common and due to edema of occipital lobe. In retina ischemia, infraction and retinal detachment can occur. The prognosis is usually good and vision return to normal following delivery.

Blood volume and coagulation:

- Haemoconcentration is a hallmark of eclampsia. Endothelial cell damage can cause activation of platelets and coagulation system leading to thrombocytopenia and DIC.

Gustaff Dekker[20]: RISK FACTOR FOR PRE-ECLAMPSIA

- Primipaternity
- Limited sperm exposure
- Pregnancy after donor insemination
- Extremes of maternal age
- Multifetal gestation
- Pre-eclampsia in previous pregnancy.
- Chronic hypertension and or renal disease.
- Maternal chronic inflammatory condition (rheumatologic disease, SLE)
- Hydropic degeneration of placenta.
- Obesity
- Thrombophilias
- Pre-eclampsia in the family

IMMINENT SYMPTOMS:

- **Headache :**

  Stefan[21] C.Kane et al 2013: The headache may be throbbing pain, needle / knife like sensation or generalized pressure. Headache has poor response to non-opoid analgesic. Transcranial Doppler Ultrasound shows strong association between abnormal cerebral perfusion pressure and headache.

- **Epigastric pain or right upper quadrant pain:**

  It frequently accompanies hepatocellular necrosis, ischemia and edema that stretch glisson capsule.

- **Nausea and Vomitting:**

  It is due to liver changes.

- **Visual disturbance:**

  Stefan[21] C.Kane et al 2013: Visual disturbance may be scotoma, photopsia, blurring vision, diplopia and amaurosis fugax. The pathology of visual disturbances were

  - Cortical blindness
  - Serous retinal detachment
  - Rare entities like purtscher like retinopathy, central retinal vein occlusion and retinal / Vitreous haemorrhages.

Oliguria:

- **Gustaaf[20] Dekker:** Oliguria or anuria may be due to combination of glomelular endotheliosis, intrarenal vasoconstriction and hypovolemia. The excess SFLT-1 play major role in glomerular endotheliosis.

Atypical pre-eclampsia /eclampsia:

- **M.Sibiai[22] et al.,** Diagnosis and management of a typical pre-eclampsia and eclampsia Am J obstet Gynecol. 2009, 200: 481e1 – 481e7.

  Classical presentation in pre-eclampsia is hypertension and proteinuria at >20wks of gestation and or <48hrs after delivery. Atypical pre-eclampsia presents without these classical finding and / or outside the time periods. They may develop before 20wks or after 48hrs postpartum and may have signs of pre-eclampsia without the usual hypertension or proteinuria.

Ayptical Pre-Eclampsia:

- Gestational hypertension plus ≥1 of following items:
  - Symptoms of Pre-eclampsia
  - Haemolysis
  - Elevated liver enzymes
  - Thrmbocytopenia.
- Gestational Proteinuria plus ≥ 1 of the following:
  - Pre-eclampsia symptoms
  - Thrmbocytopenia
  - Elevated liver enzyme

- Early sign and symptom of Pre-eclampsia– Eclampisa at less than 20 weeks of gestation. Late postpartum pre-eclampsia – eclampsia >48hours after delivery.

Signs and symptom, laboratory test results relevant to pre-eclampsia:

Sign and symptom:

- Right upper quadrant pain / epigastric pain / retrosternal pain.
- Nausea and vomiting.
- Headache
- Visual changes
- Altered mental changes
- Bleeding form Mucosal membrane.
- Jaundice.

Labaratory tests:

- Persistent proteinuria $\geq$ 300mg/24hrs.
- Platelet count < 1,00,000/mm^3
- Liver enzymes two times the upper normal limit.
- Serum creatinine > 1.2 mg/dl.
- Lactic dehydrogenase $\geq$ 2 times the upper limit of normal.

ECLAMPSIA:

Eclampsia is defined as the development of seizures that cannot be attributed to other causes and/ or unexplained coma during pregnancy or puerperium in a women with preeclampsia. About 0.5% of mild pre-eclampsia and 2.3% of severe pre-eclampsia women develop eclampsia.

Eclampsia usually occur anytime from IInd trimester to puerperium. If it occurs before 20wks, molar preganancy and antiphospholid syndrome should be considered.

Types of Eclampsia:

- Antepartum eclampsia is 38% to 53% ,
- Intrapartum eclampsia is 18% to 36%,
- Postpartum eclampsia is 11% to 24%.

Pathogenesis of seizure:

Contemporary clinical management of cerebral complications of pre-eclampsia: (Obster Gynecol Int. 2013)[21]

Two hypothesis have been proposed:

- Cerebral over-regulation resulting in vasospasm causes ischemia and intracellular (cytotoxic) edema.
- Loss of auto regulation of cerebral blood flow due to high systemic blood pressure results in hyperperfusion, endothelial damage and extracellular (vasogenic) edema.

Clinical feature:

Seizures:

- Characteristic of seizure is usually generalized, tonic – clonic seizures.
- Seizure is usually self limiting, lasts for 60-75 sec and seldom last longer than 3 – 4min.

Phase of eclamptic seizure:

Prodromal phase:

- Patient may have aura followed by convulsive movement beginning around the mouth.

Tonic phase:

- Last for 10 – 20 sec.
- Sudden loss of consciousness and posture. Eyes deviate upwards and therein brief flexion of arms followed by extension of head, neck, arms and legs.

Clonic phase:

- Lasts for 30 – 90 seconds
- There will be brief violent and generalized flexor contraction altering with progressively longer muscle relaxation.
- Tongue biting, foamy salivation, loss of bladder and bowel control can occur.
- It ends with deep inspiration.

Recovery phase:

- Convulsion subsides, respiration resumes and patient may have coma of variable duration.
- Patient has mild confusion, headache, muscle soreness and fatigue in postictal phase

Fetal:

During and immediately after an seizure fetal bradycardia occur which lasts for atleast 3 – 5 mintues. By stabilizing the mother, fetus usually recovers.

Differential Diagnosis:

- Epilepsy
- Hemorrhage or central thrombosis causing cerebrovascular accident.
- Infection like meningitis, encephalitis and cerebral malaria
- Metabolic disturbances.

Complications of eclampsia

Maternal:

- Maternal injury
- Aspiration pneumonia
- Placental abruption
- Status epileptius
- Cardio pulmonary arrest

- Acute renal failure
- HELLP Syndrome
- Retinal detachment
- Cerebral hemorrhage
- PRES syndrome
- Pulmonary edema
- Postpartum psychosis
- Coma
- Maternal death

Fetal:

- Fetal distress/bradycardia
- Hypoxic ischemic encephalopathy
- Intrauterine death

Cerebrovascular accident:

- Stroke in women with Pre-eclampsia may be either ischemic or hemorrhagic. Hemorrhagic is more common and it is the common cause of maternal death.
- Pre-eclamptic and eclamptic women with stroke has main correlation with systolic blood pressure than diastolic blood pressure.

Visual disorder:

Arias[23]: Blindness can occur in patient with severe pre-eclampsia and eclampsia, due to multiple micro hemorrhages and microinfarcts in the occipital lobe. Diplopia may occur due to functional impairment of sixth nerve palsy. Visual disturbance usually resolves after delivery.

PRES (Posterior reversible encephalopathy syndrome)

PRES is characterized by variable association of seizures activity, conscious impartment, headaches, visual abnormalities, nausea/ vomiting, and focal neurological signs. It is clinicoradiological condition diagnosed by appearance of symmetrical lesion of vasogenic edema, predominantly in parieto-occipital lobes. Coexistent ischemia / infarction has been reported due to vasoconstriction secondary to pressure from edema. PRES is not unique either to pregnancy or eclampsia and occur in any hypertensive state.

Abruption placenta :

About 7% of all patient with eclampsia have premature separation of placenta. It is often an unexpected finding at time of delivery.

Acute renal failure :

Oliguria is common in patient with severe pre-eclampsia mainly due to volume depletion which respond to fluid challenge.

In rare case oliguria may be due to ATN that occur in severe pre-eclampsia patient complicated by abruption and DIC.

Pulmonary edema:

Pulmonary edema in pre-eclampsia – eclampsia patient is common during postpartum. The cause of pulmonary edema are capillary permeability edema, cardiogenic edema or combination of the both.

HELLP Syndrome:

- HELLP syndrome was coined by Louis Weinster.
- Haemolysis, elevated liver enzyme and low platelet

Diagnostic criteria: Arias[23]

Haemolysis:

- Blurr cells, schistocytes in the peripheral smear.
- Bilirubin greater than 1.2mg/dl
- Absent plasma hepatoglobin.

Elevated liver enzyme:

- AST more than 72 Iu/l
- LDH more than 600 Iu/l

Low platelet count

- Platelet <1,00,000/mm^3

Fetal complication are

- Intrauterine fetal growth retardation
- Prematurity
- Antepartum and intrapartum asphyxia
- Intrauterine death

Investigation:

- Haematocrit, hemoglobin, peripheral smear, urine albumin, 24 hrs urinary protein, renal function test, liver function test, serum uric acid and platelet.
- If platelet count or liver enzymes are abnormal, coagulation studies and testing for hemolysis are indicated.

Proteinuria:

- Proteinuria is due to presence of capillary leak but is not a sign of renal damage.
- Change in amount does not reflect increasing severity of disease.
- 24hrs urine protein is gold standard in diagnosis of pre-eclampsia.

Uric acid measurement:

Serum uric acid is better predictor of fetal morbidity than blood pressure. The rising level is due to renal tubular function impairment and increased production secondary to tissue damage due to ischemia. Any rising level is indicative of disease progression.

Thrombocytopenia:

Platelet count falls due to endothelial activation. Fall in platelet count are associated with progressive disease and worsening outcome.

Liver function test:

The disruption of liver cells leads to release of enzyme into circulation and their level rises. AST (Asparate transaminase) is more sensitive.

Fetal Ultrasound assessment:

It helps to asses fetal growth and expected fetal weight. Doppler evaluation gives fetal well being.

Neuro-imaging:

- Cerebral edema is most common finding following eclamptic seizure.
- Intracranial hemorrhage and petechiae are major findings in the patient died due to preeclampsia

The bad prognostic factors are

- Long interval between fits and delivery
- Late referrals
- Coma
- Very high blood pressure
- Oliguria and severe proteinuria
- Abnormal liver function an HELLP syndrome.

Maternal mortality:

- Maternal morbidity due to eclampsia varies from 1 to 5%.
- Cause of death in eclampsia is
    - cerebral hemorrhage

- DIC
- Acute renal failure
- Cardio pulmonary arrest
- HELLP syndrome

Perinatal mortality:

- The perinatal mortality is due to prematurity, hypoxia and effects of drugs.

Eclampsia prophylaxis:

- 80-90% of eclampsia has prodromal symptoms.
- When patient has imminent symptoms, she should be kept in quiet room and sedation can be given if necessary.
- Antihypertensive therapy should be initiated and blood pressure to be controlled.
- Magnesium sulphate is given to prevent seizure.

Management of Eclampsia

- Clearing the airway
- Controlling seizures
- Controlling the blood pressure.
- General care and monitoring
- Delivery of the baby
- Postpartum monitoring to prevent further convulsions and other complications.

CLEARING THE AIRWAYS:

- Patient nursed in left lateral position and suction of secretions done to prevent aspiration.
- Airway or padded tongue blade placed between teeth to avoid tongue bite and maintain airway. O_2 is given at rate of 8 – 10 *l*/min.

CONTROL OF SEIZURES:

Magnesium Sulphate:

- $MgSo_4$ is drug of choice for prevention and treatment of eclampsia.
- Magnesium Sulphate is $MgSo_4.7H_2O$.
- **$MgSo_4$** It inhibits Ca^{2+} release from the intracytoplasmic storage deposits. It blocks Ca^{2+} influx through glutamate channels or through the N-methyl D-asparate receptor and suppress neuronal excitability.
- Eclamptic convulsion are prevented or arrested by plasma magnesium level of 4-7meq/*l*.
- Magnesium are mainly excreted through kidney. Magnesium intoxication is unusual when glomerular filtration rate is normal or slightly reduced.
- Pregnancy Category A.

The regimens:

Pritchard[1] Regimen.

- 4gm of Magnesium Sulphate as 20% solution intravenously at rate not exceeding 1gm/min.
- Follow promptly with 10gm of 50% $MgSo_4$ given 5gms as deep intramuscular injection in each buttock.
- Every 4 hours thereafter 5gm of 50% solution of $MgSo_4$ is injected intramuscular in alternate buttock.
- Magnesium Sulphate is given till 24hrs after delivery or after last convulsion, whichever is later after ensuring
    - Patellar reflex is brisk.
    - Respiration not depressed.
    - Urine output >100ml in previous 4hrs.

Zupan[24] regimen:

- Loading dose: 4gm of Magnesium Sulphate diluted in 100ml of fluid and given intravenously over 5 – 10min.
- Maintenance dose: 1gm per hour Magnesium Sulphate was given iv till 24 hrs after delivery or convulsion whichever is later.

Sibai[24] regimen

- Loading dose: 6gm Magnesium Sulphate diluted in 100ml of fluid and given iv over 5 – 10min.

- Maintenance dose: Magnesium Sulphate is given iv at rate of 2gm/hour.
- Magnesium Sulphate is continued till 24hrs after delivery or after convulsion whichever is later.

Monitoring during Iv infusion:

- Magnesium toxicity is monitored by
  - Assessing the deep tendon reflex periodically.
  - Serum Magnesium level can be measured at 4 – 6hrs and infusion is adjusted to maintain the level of 4 – 7 meq/l
  - Serum Magnesium level is measured if serum creatinine is $\geq$ 1.0mg/dl.

Recurrence of fit:

- If convulsion persist 15mins after initial dose give 2gm of $MgSo_4$ iv as 20% solution at a rate not to exceed 1gm/min.

Side effects :

Minor side effects – feeling of warmth, flushing, somnolence, nausea and vomiting, muscle weakness.

Major side effects – loss of patellar reflex and respiratory arrest. It is rare.

- Loss of patellar reflex occurs when Serum Magnesium level 8 -10meq/d*l.*
- Respiratory depression occurs when Serum Magnesium level is >13meq/*l.*

Magnesium toxicity:

- Calcium gluconate, 10ml of 10% solution given iv over 3 minutes to reverse the magnesium toxicity.

Absolute contraindication for $MgSo_4$:

- Myasthenia gravis
- Myocardial infraction.

- **The MAGPIE[1] trial** was a large multicentric randomised trial, proved beyond doubt the effectiveness of magnesium sulphate in proplylaxis and its safety.
- **Simth[25] JM et al.,**: The study is done for the conformation of safety profile of $MgSo_4$. The side effects were found at low rate. In their study the side effect of absent patellar reflex is 1.6%, respiratory depression is1.3% and use of Calcium gluonate to reverse the effect of $MgSo_4$ is <0.2%.
- **The Multinational Eclampsia Trial Collaborative Group Study (1995)[1]** Magnesium Sulphate has lower incidence of recurrent seizure and maternal death rate compared to phenytoin and diazepam. Magnesium Sulphate is more effective in controlling seizure than lytic cocktail.
- **Baha[26] M.Sibai et al:** $MgSo_4$ is equally effective in preventing recurrent seizure in all the three regimen (Pitchard, Zupan and Sibai)

- **Singh[24] et al:** Magnesium sulphate given by Pitchard and Sibai regimen was 100% effective in controlling recurrent seizure and 98% in Zupan regimen.

Maternal consideration:

When serum magnesium level is relatively high myometrial contractility is depressed. However the therapeutic dose given has no effect on myometrial contractility. **Levno[27] and colleagues (1998)** showed $MgSo_4$ did not alter the need for oxytocin stimulation of labour, admission-delivery interval and mode of delivery. For inhibition of uterine contractility at least serum magnesium level should be 8 to 10 meq/*l.*

Fetal consideration:

- When parenterally administered it crosses the placenta and increase the fetal level. It remains controversial whether $MgSo_4$ reduces fetal heart variability and reactivity.
- **Williams[1]:** $MgSo_4$ has Protective effect against cerebral palsy in very low-birth weight infants.

Phenytoin:

It was synthesized in 1908 as a barbiturate analogue.

Mechanism:

By prolonging the inactivated state of voltage sensitive neuronal Na^+ channel, phenytoin has stabilising influence on neuronal membrane.

Thus prevents repetitive detonation of normal brain cells during depolarization shift that occur in epileptic patient.

Maternal consideration:

Clearance is increased during pregnancy, dose adjustment should be based on clinical symptom and not solely on serum drug concentration.

Side effects:

Gingival hyperplasia, Megaloblastic anaemia, Hirsutism, Osteomalacia Leucopenia, Agranulocytosis, Exfolative dermatitis, Polyarteritis nodosa, Steven Johnson Syndrome, SLE.

Fetal consideration:

Phenytoin crosses the human placenta. If given in 1^{st} trimester causes fetal hydantion syndrome – hypoplastic phalangs, microcephaly, cleft palate and hare lip.

Control of Blood pressure:

Labetalol:

- Adrenergic antagonist. Selective $\propto$-1 and non-selective β-1 and β-2 adrenergic receptor antagonist.

Dose: Labetolol-200-400mg/day in 2 divided doses.

Pregnancy Category C.

Labetolol is considered as drug of choice to control blood pressure (NICE guidelines).

Maternal Consideration:

- It reduces BP more slowly than nifedipine and cardiac index is not altered. It is drug of choice for hypertensive women with tachycardia. It reduces cerebral pressure without altering cerebral perfusion.

Side effects:

- Hepatic necrosis, SLE, bronchospasm, dizziness, nausea and vomiting, fatigue, dyspepsia, rhinitis, edema and postural hypotension.

Fetal consideration:

- Labetalol crosses human placenta. Doppler flow studied reveal no change in umbilical, uterine and middle cerebral resistance.

Nifedipine:

- Calcium channel blocker
- Dose: 10-20mg three to four times daily, 10-20mg one or twice daily in extended release tablet
- Maximum dose that can be given is 120mg/day.

Pregnancy Category C

Maternal Consideration:

- Proven safe and effective. In maternal cerebral blood flow there is reduction in middle cerebral artery flow velocities but there is no change in uteroplacnetal flow.

Fetal consideration:

- Nifidipine crosses the human placenta. Newborn exposed to nifedipine has lower NICU admission rates, lower incidences of RDS, intracranial bleeding and neonatal jaundice.

Obstetric Management:

- Termination of pregnancy is cure for Imminent eclampsia and eclampsia.
- The mode of delivery depends on gestational age, fetal presentation and cervical scoring.
- Timing of delivery affects the outcome of both mother and baby. A rushed delivery in unstable patient and delay in delivery in sick patient may cause maternal mortality.
- Vaginal delivery is preferred route after eclamptic seizure. Following seizure labour starts spontaneously or can be induced to improve Bishop Score. Prolonged induction is however be avoided.
- In current obstetrical practice the majority of eclamptic women are delivered by cesarean section. Liberalization of cesarean section for eclamptic women is done inorder to reduce the maternal morbidity. Cesarean is done for term fetus with unfavourable cervix, poor progression of labour and fetal distress..
- Anesthesia of choice is regional, spinal or epidural .

- In postpartum period first 24 - 48hrs is crucial as BP fluctuate and patient is at risk of developing complication.
- Antihypertensive is continued throughout the labour and required for 24 - 48hrs and need to be gradually decreased.

Postnatal assessment:

- If hypertension or proteinuria persist after 6weeks postpartum, it is necessary to investigate further.
- It is important to counsel patient for early booking in future pregnancies.
- Long term prognosis and counselling for future pregnancies (including contraception) should be discussed.

MATERIALS AND METHODS

MATERIALS AND METHODS

The study was carried out in the department of Obstetrics and Gynaecology, Government Theni Medical College during the period of August 2013 to July 2014. Prospective study was conducted on patient admitted for imminent eclampsia and eclampsia.

102 Antenatal mother were selected for the study. 53 of them were imminent eclampsia patient and 49 were eclampsia patient.

The purpose of this study is to analyse the determinants of imminent eclampsia and eclampsia like age, parity, socio economic status, booking status, referral status, parity, gestational age, prodromal symptoms, blood pressure, convulsion details, mode of delivery and maternal complication.

Inclusion Criteria:

1) ALL PREGNANT WOMEN WITH IMMINENT ECLAMPSIA
2) ALL PREGNANT WOMEN WITH ECLAMPSIA INCLUDING
    - Antepartum eclampsia.
    - Intrapartum eclampsia.
    - Postpartum eclampsia.

Exclusion criteria:

Other causes of convulsion in pregnancy

- Epilepsy
- Trauma (head injury)
- Metabolic disorder (anaemia, electrolyte imbalance, hepatic / renal failure, hypogylcemia)
- Poisoning (Strychnine, CNS stimulant)
- Infection (Meningitis, Encephalitis, Cerebral Malaria)
- Functional
- Brain tumours.

History:

History is collected from patient and if patient is unconscious or in postictal state, history is collected from attender. A detailed history of the patient regarding age, parity, socio-economic status, booking status, gestational age, prodromal symptom, details about convulsion, referral and treatment (including $MgSo_4$ administration) at referral centre were noted down. Details about previous obstetric history were recorded.

Emergency care:

When the patient with convulsion is received, Patient nursed in left lateral position and suction of secretions done to prevent aspiration. The bedside rails is elevated to prevent maternal injury. Mouth gag is placed to

prevent tongue bite. Oxygen by mask is given at rate of 8-10 litre/minute. IV lifeline started.

Clinical evaluation:

Detailed general examination and obstetric examination were done. On general examination conscious level, pedal edema, anaemia, jaundice, BMI, Blood pressure, pulse rate, respiratory rate, temperature, fundus and nature of fits were recorded. RS, CVS, CNS, examined.

For all cases antihypertensive given. T.Labetalol 100mg bd is started and dose adjusted according to BP. If diastolic BP is >110mmHg. T.nifedipine 10mg bd started. For patient who had uncontrolled BP, IV labetalol given. Dose of IV labetalol is 20mg IV bolus given initially. If the blood pressure does not decrease to the expected range (80-110 diastolic) in 10 minutes, additional dose of 40mg, then 80mg may be administered every 10 minutes as needed to a maximum of 300mg. Once the blood pressure is in adequate range, oral labetalol 200 – 400mg every 12hours is started. For continuous IV administration, one 40-ml vial containing 200mg labetalol is added to 160ml of lactated Ringers solution. The resultant solution will contain 1mg/ml. the initial dose is 20mg/hour. This dose can be doubled every 20minutes up to a maximum of 200mg/hour. The therapeutic range is usually between 50 and 200mg/hour. Once the blood pressure reaches the

desired level, the IV solution is discontinued and the patient started an oral labetalol, 100-400mg every 6 -12 hours.

Loading dose $MgSo_4$ given as therapeutic in all eclampsia and Prophylatic in all imminent eclampsia. $MgSo_4$ was given according to Pritchard regimen.

Pritchard regimen:

Loading dose: 4gm of 20% Magnesium sulphate is given iv slowly over 5minutes. 10gm of 50% solution given intramusculary as 5gm in each buttock.

Maintenance dose: 5gm of 50% solution of Magnesium sulphate given every 4hours. Magnesium sulphate is continued for 24hours after delivery or convulsion whichever is later.

While giving repeat dose of Magnesium sulphate following are monitored

- Respiratory rate
- Patellar reflex
- Urinary output

Repeat dose of Magnesium sulphate in withheld if:

- Respiratory rate is <16 per minute.
- Patellar reflex are absent
- Urinary output is <30ml per hour over preceding 4 hours.

Bladder was catheterized and hourly urine output monitored. Patellar reflex and respiratory rate were monitored. Half hourly PTR chart maintained and BP recorded second hourly and whenever necessary.

Obstetric Management:

After stabilizing the patient, mode of delivery was planned according to gestational age, presentation, viability of fetus, cervical scoring and pelvic assessment.

Vaginal delivery is preferred and induction done with PGE2 gel for patient who was not in labour. When patient is in labour, acceleration is done with aminotomy and oxytocin infusion. LSCS is done when there is maternal, fetal indication and when PGE2 induction failed.

Immediately after delivery of baby 10 units of oxytocin given intramasucular. The patient was carefully monitored anticipating complication like postpartum hemorrhage and postpartum ecalmpsia.

The patient was observed for 48 hours in Eclampsia room. Blood pressure was monitored and antihypertensive continued. $MgSo_4$ was continued 4^{th} hourly till 24hours after delivery by monitoring patellar reflex, respiratory rate and urine output. After 48hours patient shifted to postnatal or immediate postoperative ward respectively.

Neonate:

Details of baby such as birthweight, maturity, sex and complication like prematurity, IUGR, birth asphyxia and mortality were recorded. Baby was followed till discharge.

Postpartum care:

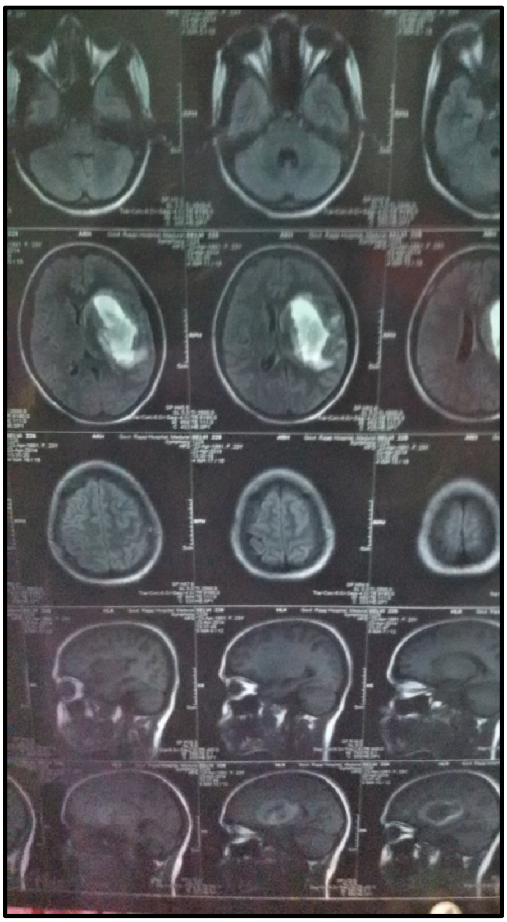
Postnatally antihypertensive drugs continued and slowly tapered when blood pressure return to normal. If BP doesnot return to normal patient was evaluated for other medical condition causing elevation of blood pressure. While discharge patient was advised for contraception and review after a week. Patient was advised about early booking in next pregnancy.

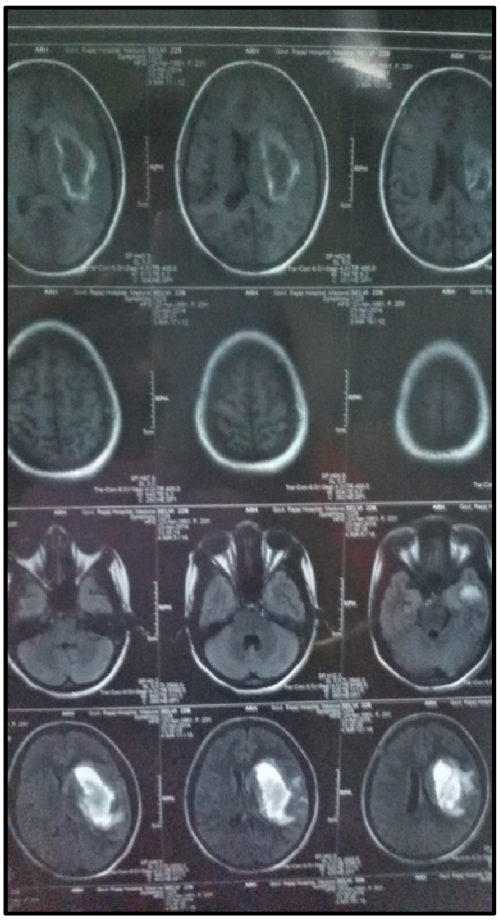
ECLAMPSIA PATIENT

PRETERM BABY

MRI OF ECLAMPSIA PATIENT

WITH CEREBRAL HEMORRHAGIC INFARCT





OBSERVATIONS & RESULTS

For period of 1 year from August 2013 to July 2014

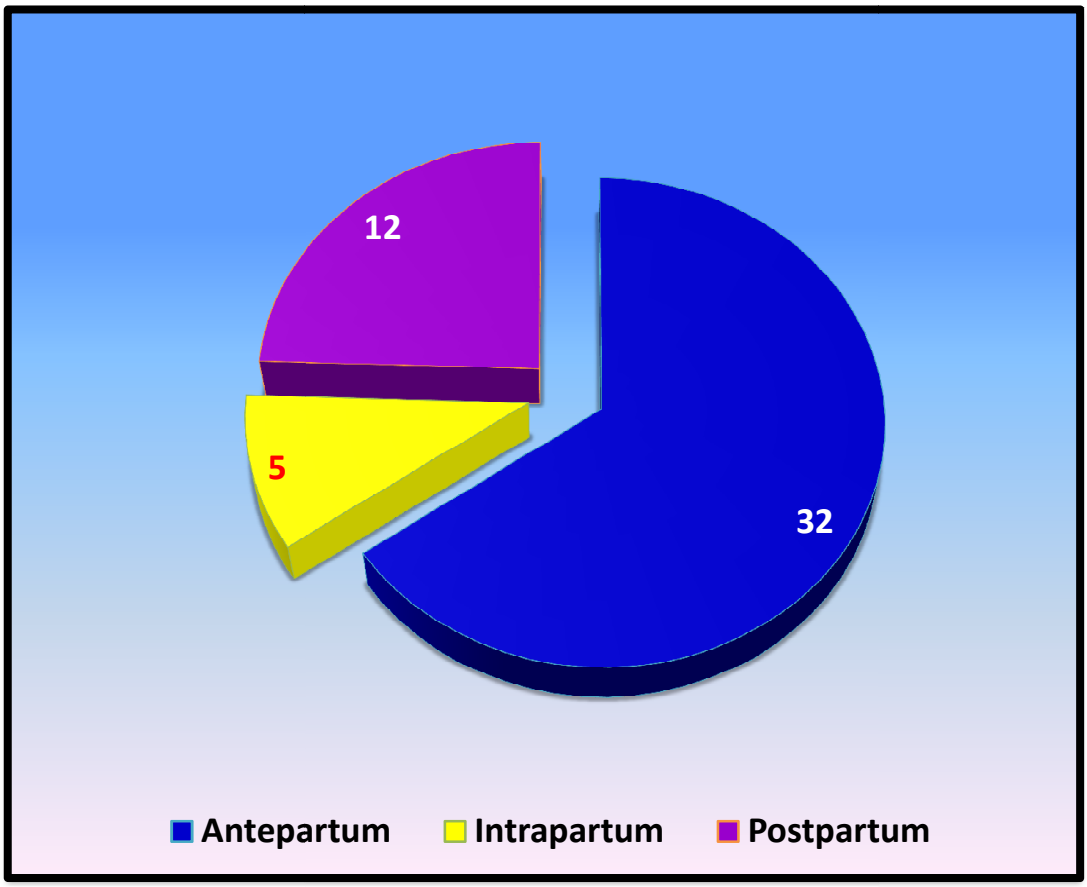
Total number of deliveries during the period of August 2013 to July 2014 in our hospital is 5420

Incidence of Imminent eclampsia and eclampsia

Total number of deliveries	**5420**	
Type of eclampsia	**No.**	**Incidence**
Imminent eclampsia	53	0.97%
Eclampsia	49	0.90%

- Incidence of Imminent eclampsia is 0.97%
- Incidence of Eclampsia is 0.90%

TYPES OF ECLAMPSIA

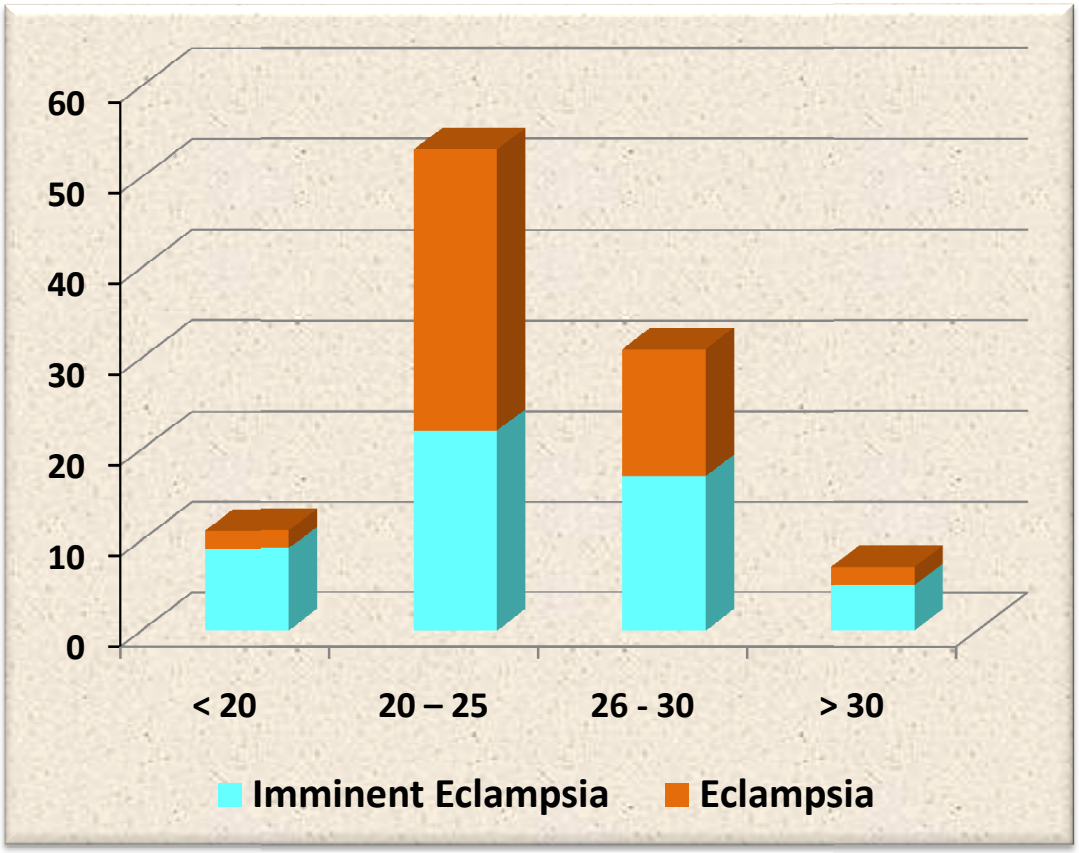


TYPES OF ECLAMPSIA

Type	Number	Percentage
Antepartum	32	65.31%
Intrapartum	5	10.20%
Postpartum	12	24.48%

- Antepartum eclampsia is 65.31%
- Intrapartum eclampsia is 10.20%
- Postpartum eclampsia is 24.48%

AGE

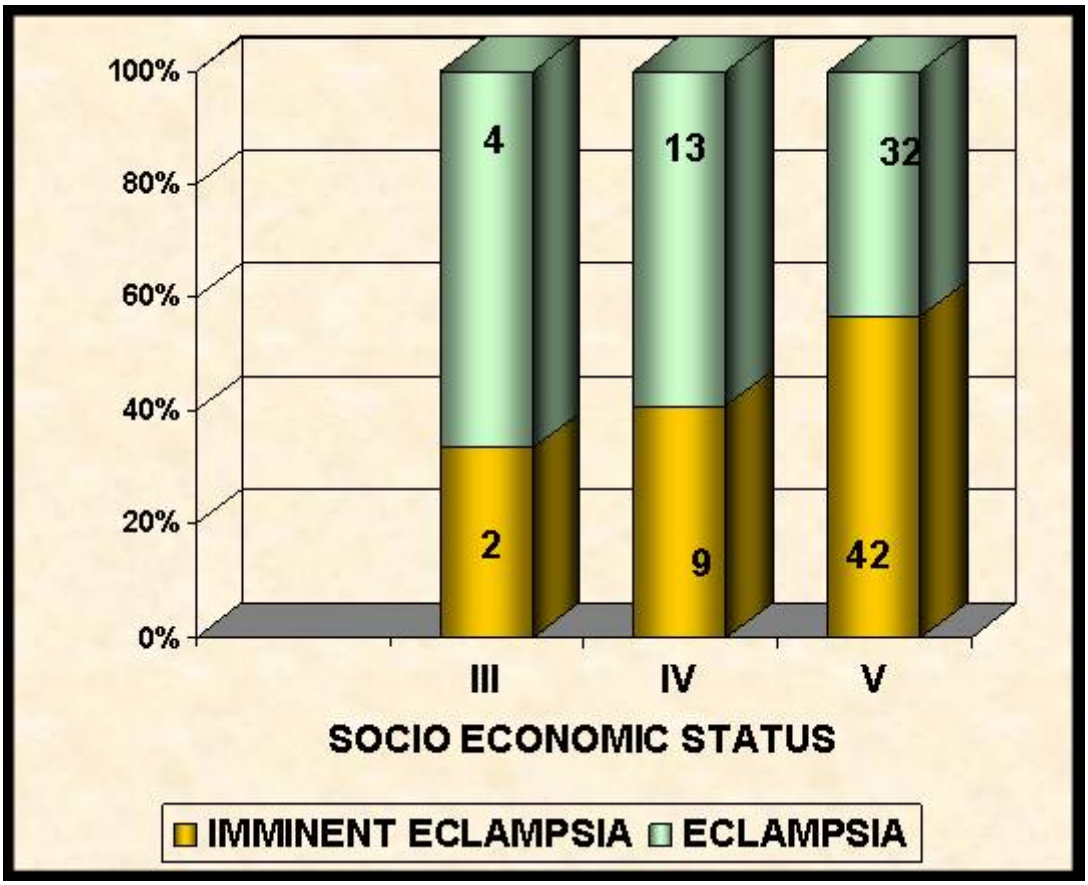

60
50
40
30
20
10
0
< 20
20 – 25
26 - 30
> 30
Imminent Eclampsia
Eclampsia

AGE

Age	Imminent Eclampsia	%	Eclampsia	%
< 20	9	16.9	2	4.1
20 – 25	22	41.5	31	63.3
26 - 30	17	32.07	14	28.6
> 30	5	9.43	2	4.1

- 22 out 53 patient of imminent eclampsia were in the age group of 20 – 25 years.
- 31 out of 49 patient of eclampsia were in the age group of 20 – 25 years.

SOCIO ECONOMIC STATUS

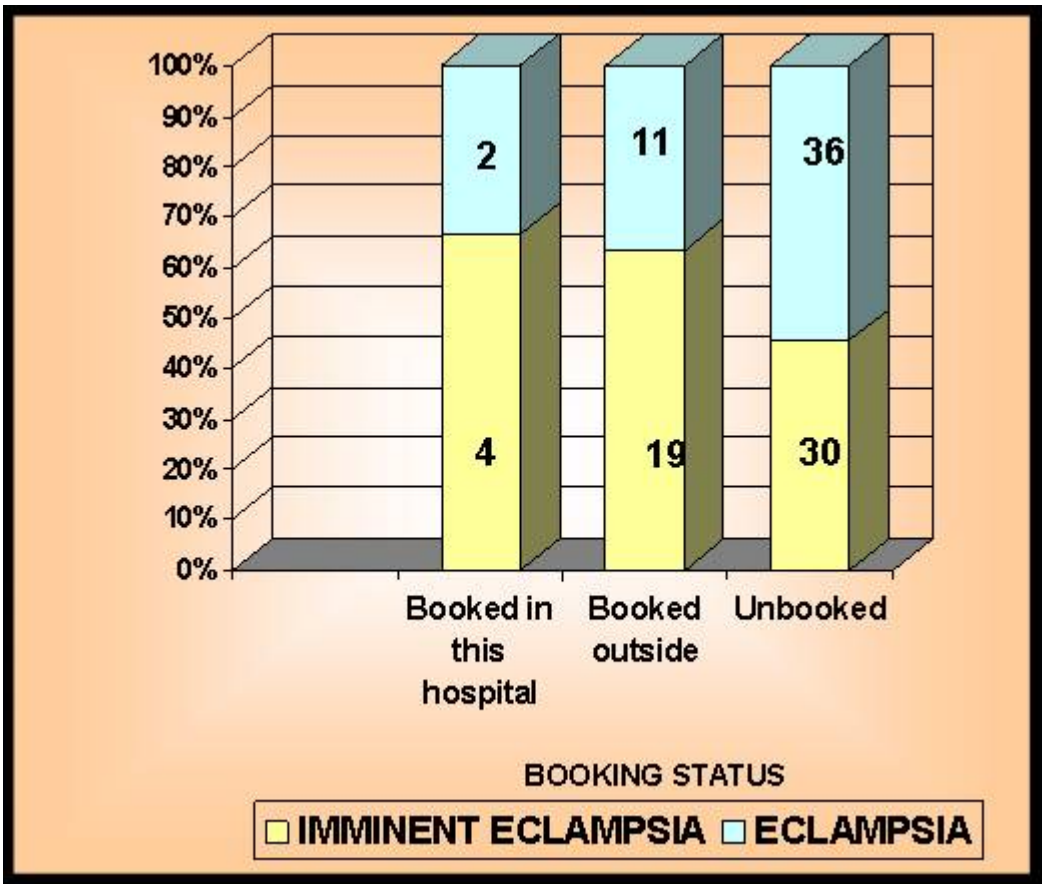


SOCIO ECONOMIC STATUS

Socio economic status	Imminent Eclampsia	%	Eclampsia	%
I	-	-	-	-
II	-	-	-	-
III	2	3.77	4	8.16
IV	9	16.98	13	26.53
V	42	79.25	32	65.30

- 42 out of 53 patient of imminent eclampsia belongs to class V economic status.
- 32 out of 49 patient of eclampsia belongs to class V economic status.

BOOKING STATUS

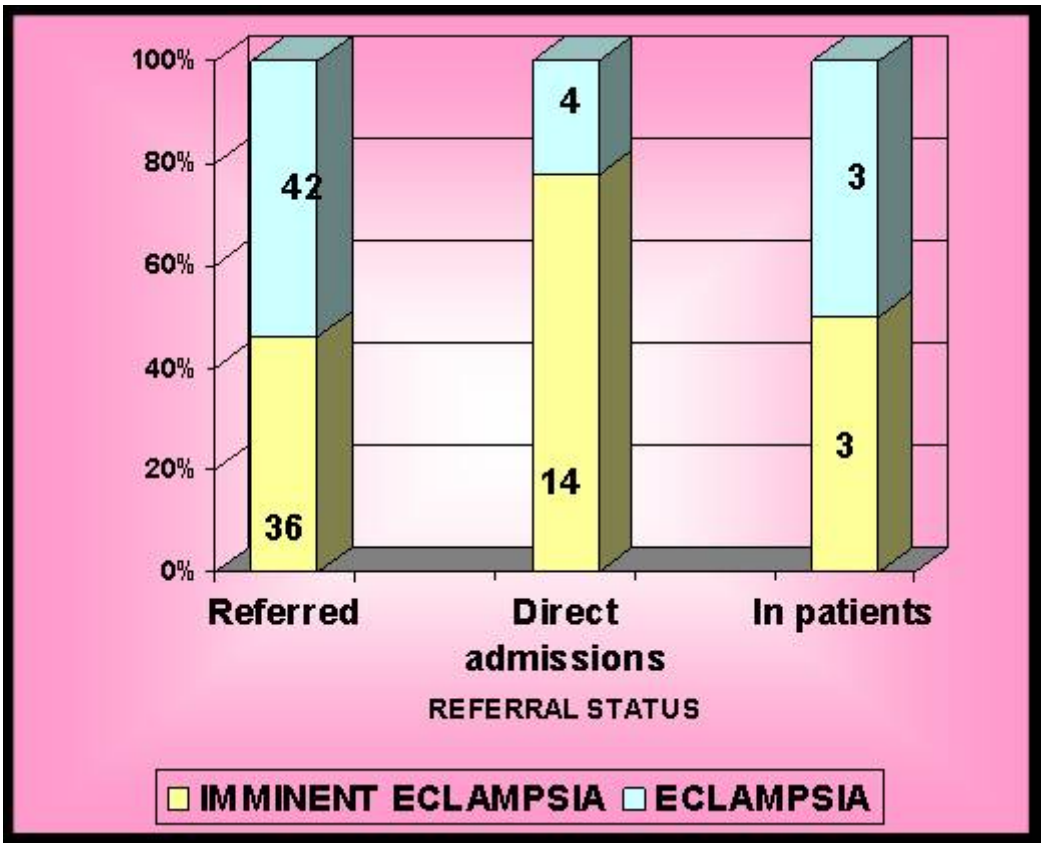


BOOKING STATUS

Booking Status	Imminent Eclampsia	%	Eclampsia	%
Booking in our hospital	4	7.55	2	4.08
Booked outside	19	35.84	11	22.44
Unbooked	30	56.6	36	73.46

- 30 out of 53 patient in imminent eclampsia were unbooked and 36 out of 49 patient in eclampsia group were unbooked.

REFERRAL DETAILS



REFERRAL DETAILS

Referral details	Imminent Eclampsia		Eclampsia	
	No.	%	No.	%
Total no. of referred in cases	36	67.9	42	85.71
Direct admission	14	26.4	4	8.16
Inpatient	3	5.6	3	6.12

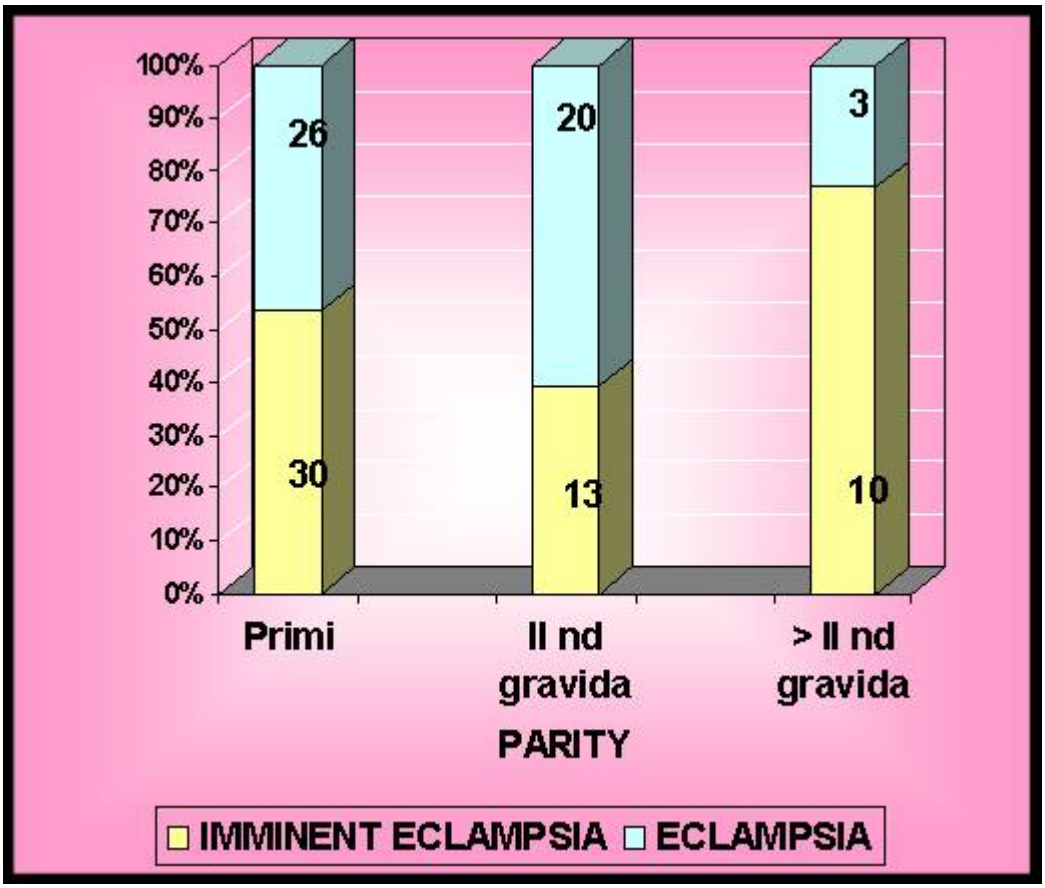
- 42 out of 49 eclampsia patient were referred to our hospital, four patient of eclampsia were admitted directly in our hospital and three inpatient had convulsion. 36 cases of imminent eclampsia were referred to our hospital, 3 patient of pre-eclampsia in ward developed imminent symptom. 14 cases of imminent eclampsia were admitted directly.

Loading dose $MgSo_4$ received before admission in first referral unit:

Types of Eclampsia	Imminent Eclampsia		Eclampsia	
	No.	%	No.	%
No. of Patient received $MgSo_4$	12	23.08	41	83.67

- 23.08% of imminent eclampsia patient and 83.67% of eclampsia patient received loading dose $MgSo_4$ in the referring unit.

PARITY

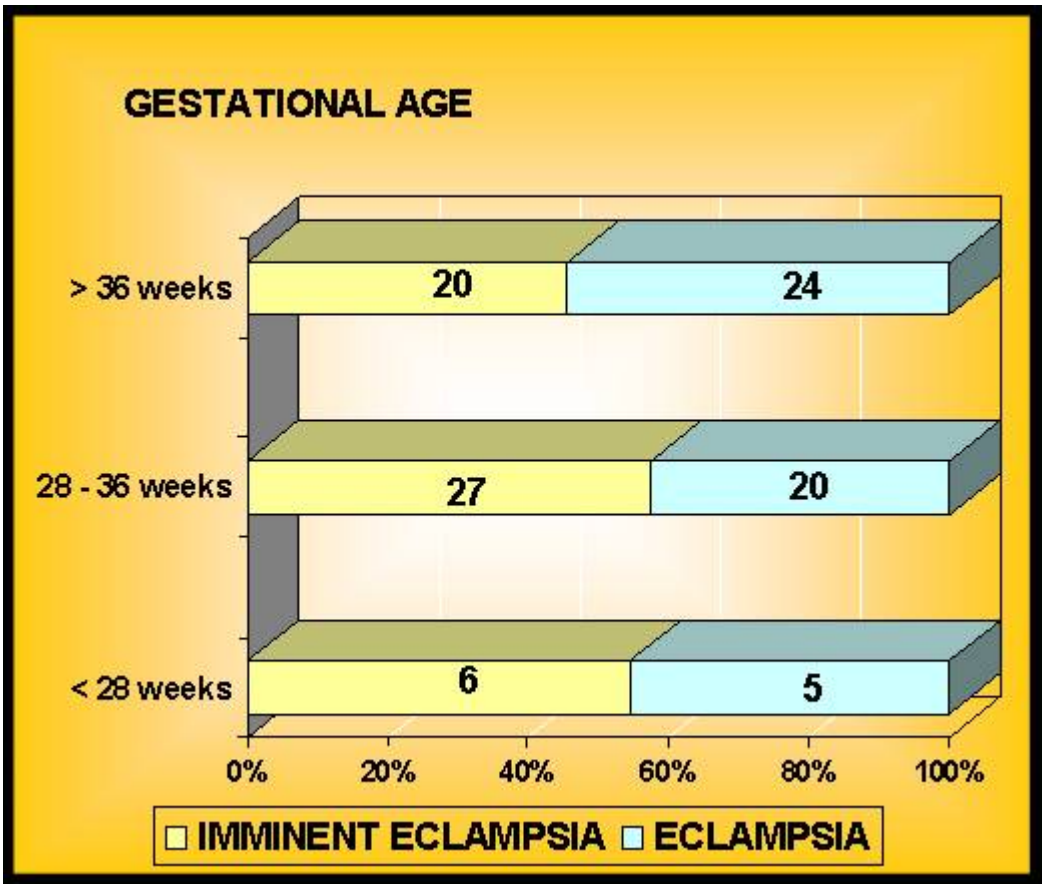

100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
26
30
20
13
3
10
Primi
II nd gravida
> II nd gravida
PARITY
IMMINENT ECLAMPSIA
ECLAMPSIA

PARITY

Parity	Imminent Eclampsia	%	Eclampsia	%
Primi	30	56.6	26	53.06
IInd gravid	13	24.52	20	40.82
> IInd gravid	10	18.87	3	6.12

- 30 out of 53 imminent eclampsia patient were primi.
- 26 out of 49 in eclampsia patient were primi.

GESTATIONAL AGE

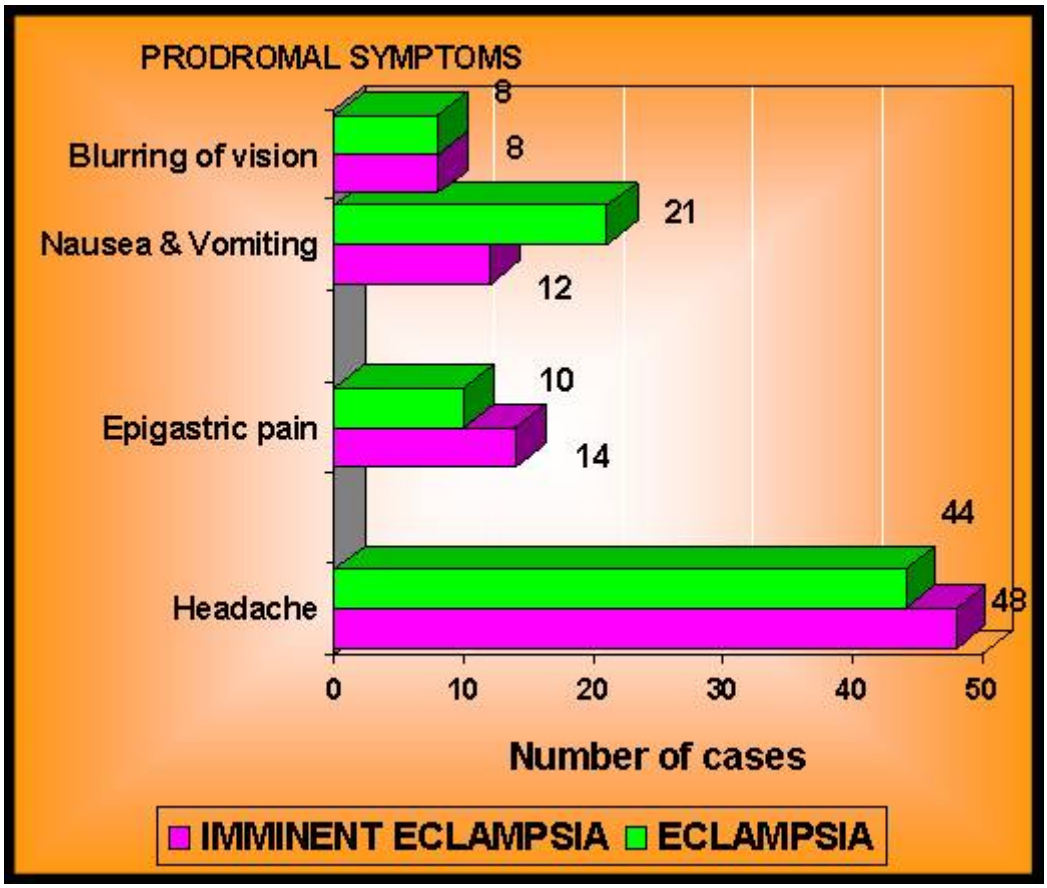


GESTATIONAL AGE

Gestational Age	Imminent Eclampsia	%	Eclampsia	%
< 28 wks	6	11.32	5	10.20
28- 36 wks	27	50.94	20	40.81
≥ 37 wks	20	37.73	24	48.97

- 27 out of 53 imminent eclamptic patient had gestational age of 28 - 36 weeks.
- 20 out of 49 eclamptic patient had gestational age between 28 - 36 weeks.
- 20 out of 53 imminent eclamptic patient had gestational age of ≥ 37 weeks.
- 24 out of 49 eclampsia patient were above 37wks of gestational age.

PRODROMAL SYMPTOMS

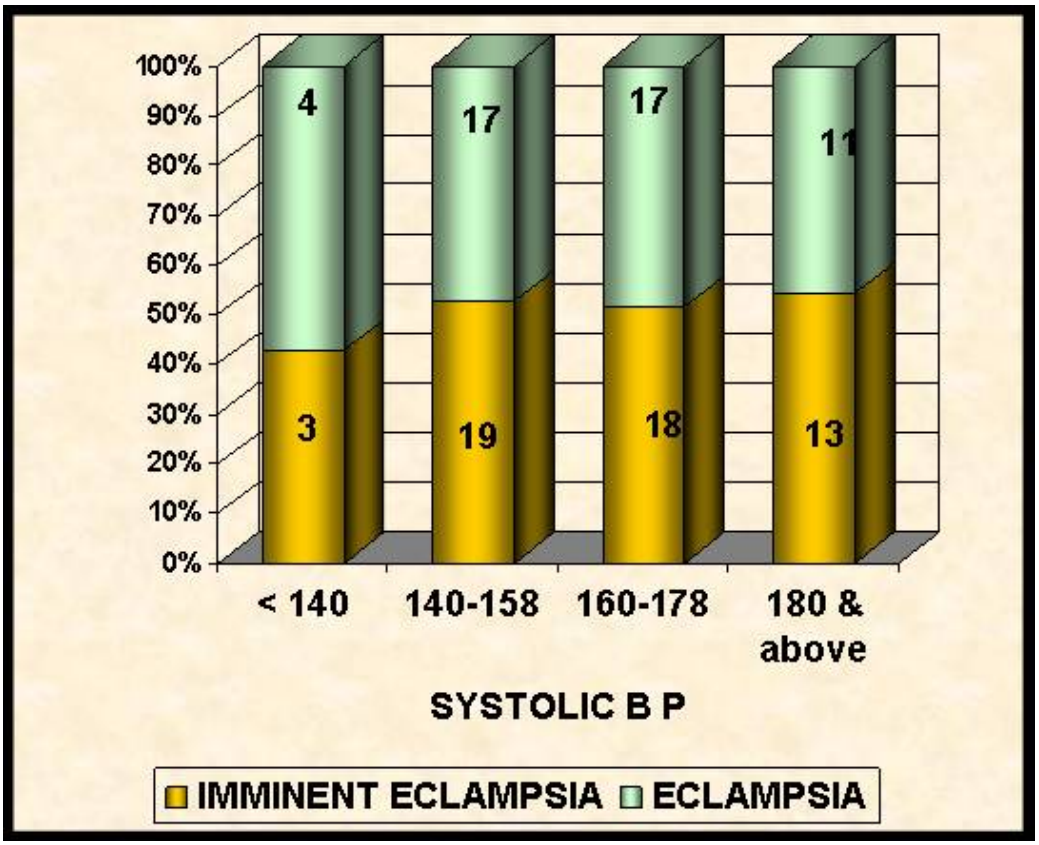


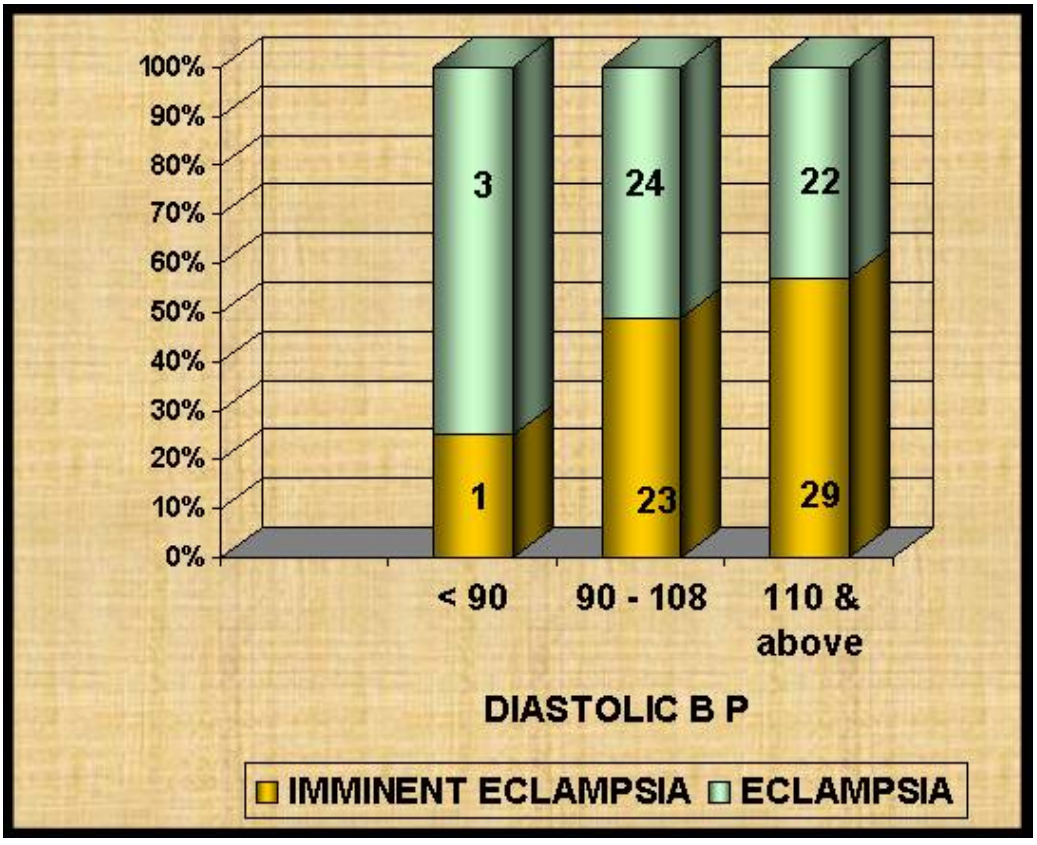
PRODROMAL SYMPTOMS

Prodromal symptoms	Imminent Eclampsia	%	Eclampsia	%
Headache	48	90.57	44	89.79
Epigastric pain	14	26.4	10	20.4
Nausea & vomiting	12	22.64	21	42.85
Blurring of vision	8	15.09	8	16.33

- 48 out of 53 patient of imminent eclampsia had headache.
- 8 out of 53 patient of imminent eclampsia had blurring of vision.
- 44 out of 49 patient of eclampsia had headache.
- 8 out of 49 patient of eclampsia had blurring of vision.

BP AT THE TIME OF ADMISSION





BP AT THE TIME OF ADMISSION

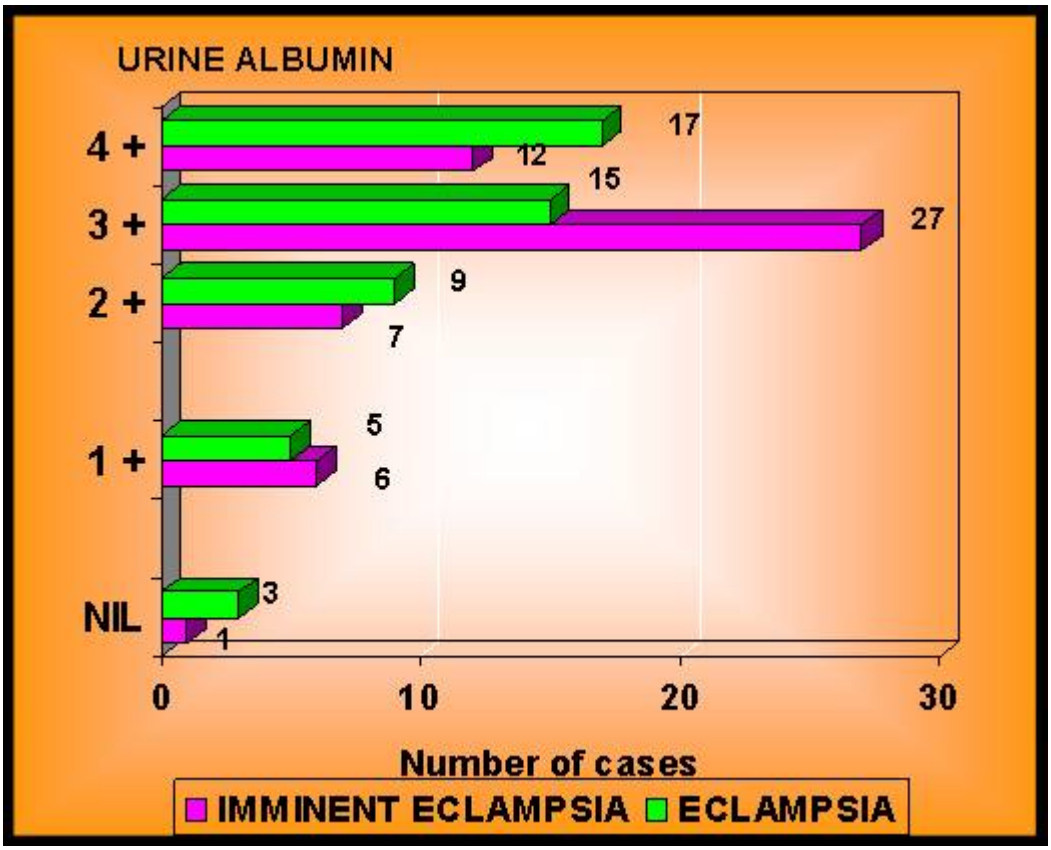
BP		Imminent Eclampsia	%	Eclampsia	%
Systollic	<140	3	5.66	4	8.16
	140-158	19	35.85	17	34.69
	160-178	18	33.96	17	34.69
	≥ 180	13	25.0	11	22.45
Diastollic	<90	1	1.88	3	6.12
	90-108	23	43.4	24	48.98
	≥ 110	29	54.7	22	44.9

- 29 out 53 patient of imminent eclampsia had diastolic BP >110mmHg
- 22 out 49 patient of eclampsia had diastolic BP >110mmHg.
- 31 out 53 patient of imminent eclampsia had systolic BP >160mmHg.
- 28 out 49 patient of eclampsia had systolic BP >160mmHg.

Out of 53 patient of Imminent eclampsia one patient had BP 130/80mmHg.

Out of 49 patient of eclampsia three patient had BP <140/90mmHg.

URINE ALBUMIN



URINE ALBUMIN

Urine Albumin	Imminent Eclampsia	%	Eclampsia	%
Nil	1	1.88	3	6.12
1+	6	11.33	5	10.2
2+	7	13.2	9	18.36
3+	27	50.94	15	30.61
4+	12	22.64	17	34.7

- 27 out 53 patient of imminent eclampsia had 3+ albuminuria.
- 17 out 49 patient of eclampsia had 4+ albuminuria.
- 1 out 53 patient of imminent eclampsia had no albuminuria.
- 3 out 49 patient of eclampsia had no albuminuria.

NO. OF CONVULSION

No. of convulsions	Eclampsia	%
1	19	38.78
2 – 5	26	53.06
6 – 10	3	6.1
More than 10	1	2.04

- 19 out 49 patient of eclampsia had 1 convulsion.
- 26 out 49 patient of eclampsia had 2 - 5 convulsions.
- 3 out 49 patient of eclampsia had 6 – 10 convulsions.
- 1 out 49 patient of eclampsia had more than10 fits (status epileptics)

ADMISSION DELIVERY INTERVAL

	Average time
Imminent Eclampsia	6.15 hours
Eclampsia	4.50 hours

- In our study average admission delivery interval was 6.15 hours for imminent eclampsia and 4.50 hours for eclampsia patients

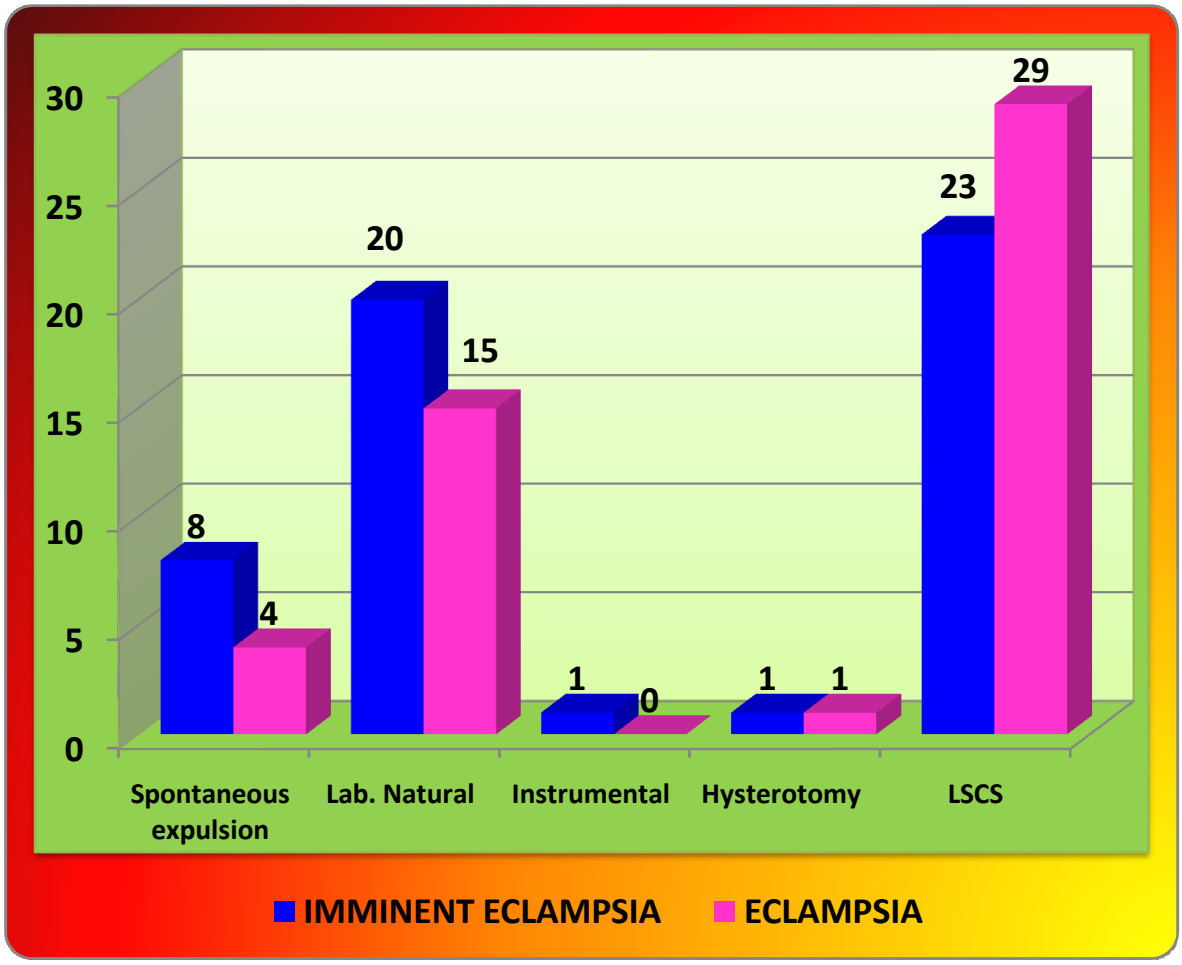
Convulsion delivery interval

Convulsion delivery interval	**AP+ IP n = 32 + 5 = 37**	**Perinatal Mortality**		**Maternal Mortality**	
		n = 40	**%**		**%**
≤ 6 hrs	12	1	2.5	-	-
7 – 12 hrs	19	3	7.5	-	-
13 – 24 hrs	6	4	10.0	2	5.4
> 24 hrs	-	-	-	-	-

* there are two twins in antepartum and one twin in intrapatrum eclampsia patient.

- Eclampsia patient who delivered within 6hrs has 2.5% of perinatal death and no adverse maternal outcome.
- Eclampsia patient who delivered within 13 - 24hrs has 10.0% of perinatal death and 5.4% of maternal death.

MODE OF DELIVERY

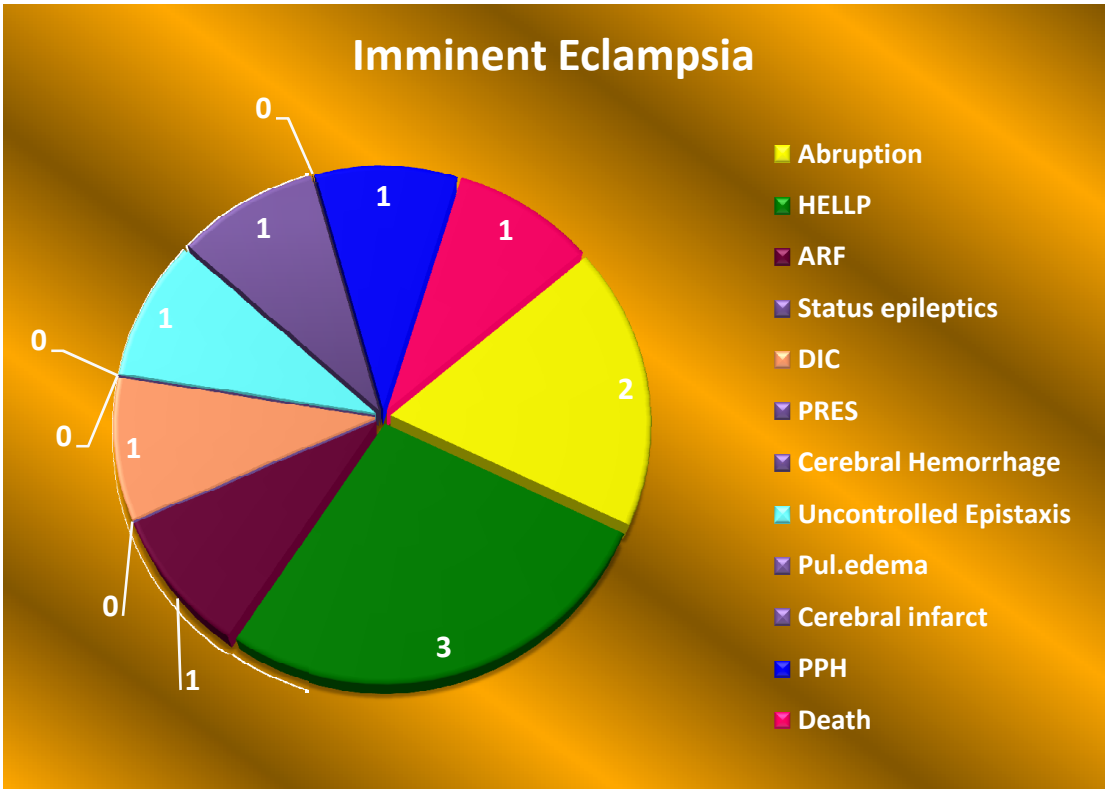


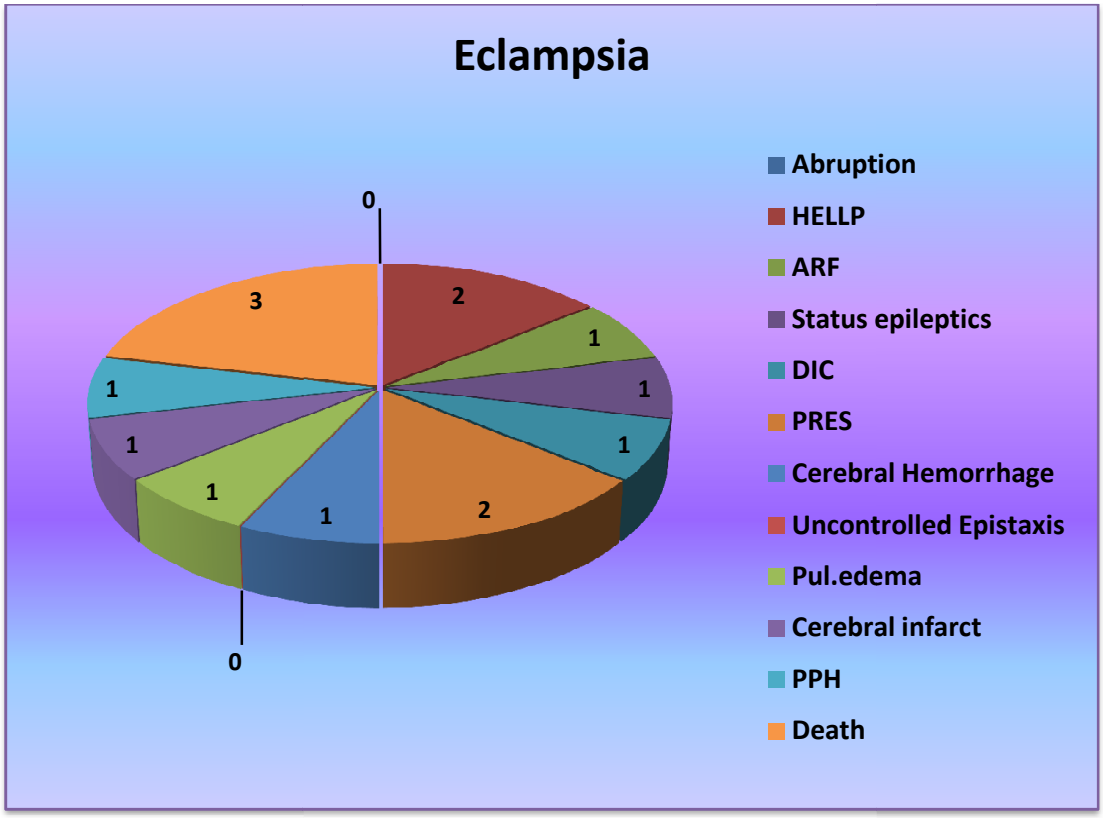
MODE OF DELIVERY

Mode of delivery	Imminent Eclampsia	%	Eclampsia	%
Spontaneous expulsion	8	15.09	4	8.16
Lab. Natural	20	37.73	15	30.61
Instrumental	1	1.88	-	-
Hysterotomy	1	1.88	1	2.04
LSCS	23	43.40	29	59.18

- Out of 53 patient of Imminent eclampsia 23 patient delivered by caesarian section and 20 patient delivered by labour natural.
- Out of 49 patient of eclampsia, 29 patients delivered by caesarian section and 15 patient delivered by labour natural.

MATERNAL COMPLICATION





MATERNAL COMPLICATION

Maternal complication	Imminent Eclampsia	%	Eclampsia	%
Abruption	2	3.72	-	-
HELLP	3	5.66	2	4.08
ARF	1	1.88	1	2.04
Status epileptics	-	-	1	2.04
DIC	1	1.88	1	2.04
PRES	-	-	2	4.08
Cerebral Hemorrhage	-	-	1	2.04
Uncontrolled Epistaxis	1	1.8	-	-
Pul.edema	1	1.8	1	2.04
Cerebral infarct	-	-	1	2.04
PPH	1	1.8	1	2.04
Death	1	1.8	3	6.12

- 3 patient of Imminent elampsia and 2 patient of ecalmpsia had HELLP syndrome.
- 1 patient of eclampsia had status epilepticus.
- 2 patient of eclampsia had PRES syndrome.
- 1 patient of eclampsia had cerebral infaract.

- Maternal death in Imminent eclampsia is one.
    - 1 patient of Imminent eclampsia died due to pulmonary embolism.
- Maternal death in eclampsia is three.
    - Maternal death in Antepartum eclampsia is two and in Postpartum eclampsia is one.
    - 1 patient of Antepartum eclampsia died due to HELLP syndrome due to hepatic repture.
    - 1 patient of Antepartum eclampsia had cerebral hemorrhage and died.
    - 1 patient of Postpartum eclampsia died due to encephacopathy.

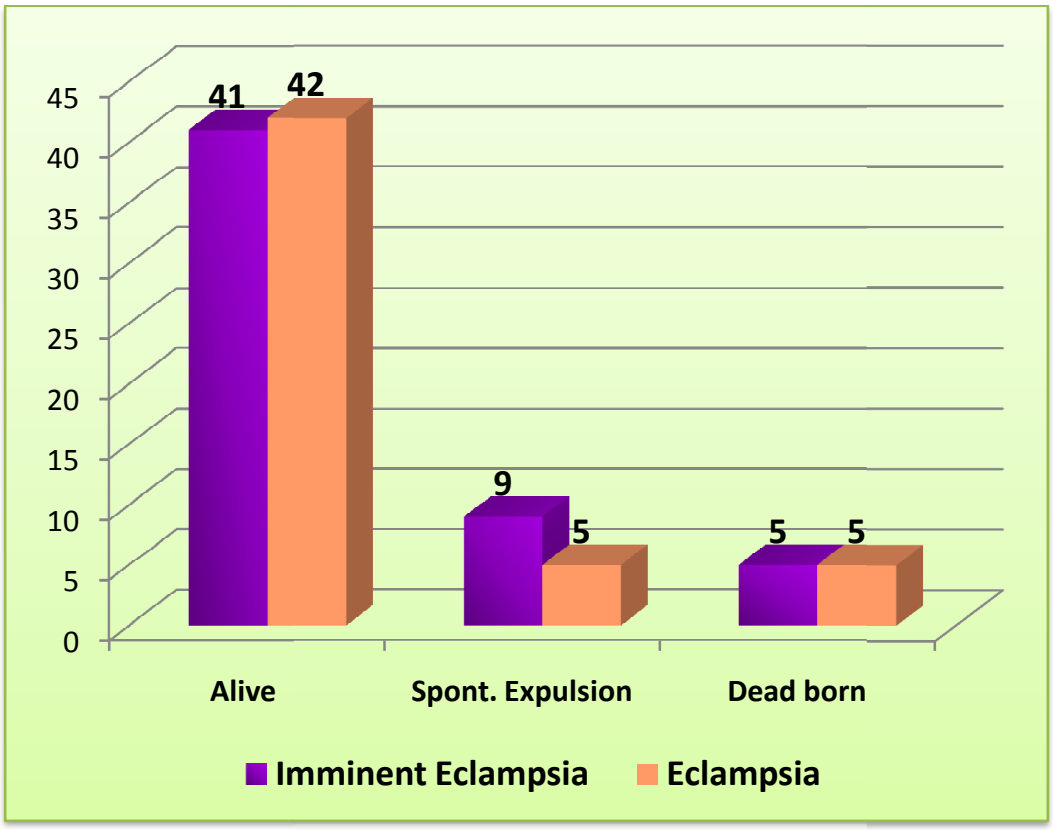
BIRTH WEIGHT

Birth Weight kg	Imminent Eclampsia	%	Eclampsia	%
< 1 kg	9	16.36	5	9.61
1 – 1.5	16	29.09	7	13.46
1.6 – 2	13	23.63	15	28.84
2.1 – 2.5	9	16.36	16	30.76
> 2.5	8	14.54	9	17.30

***** there are 2 twins in imminent eclampsia and 3 twin in eclampsia group.

- 8 out of 55 babies delivered by imminent eclampsia patient had birth weight >2.5kg.
- 9 out of 52 babies delivered by eclampsia patient had birth weight >2.5kg.
- 16.36% babies of Imminent eclampsia group and 30.76% babies of eclampsia group had birth weight between 2.1 - 2.5kg.

NEONATAL OUTCOME



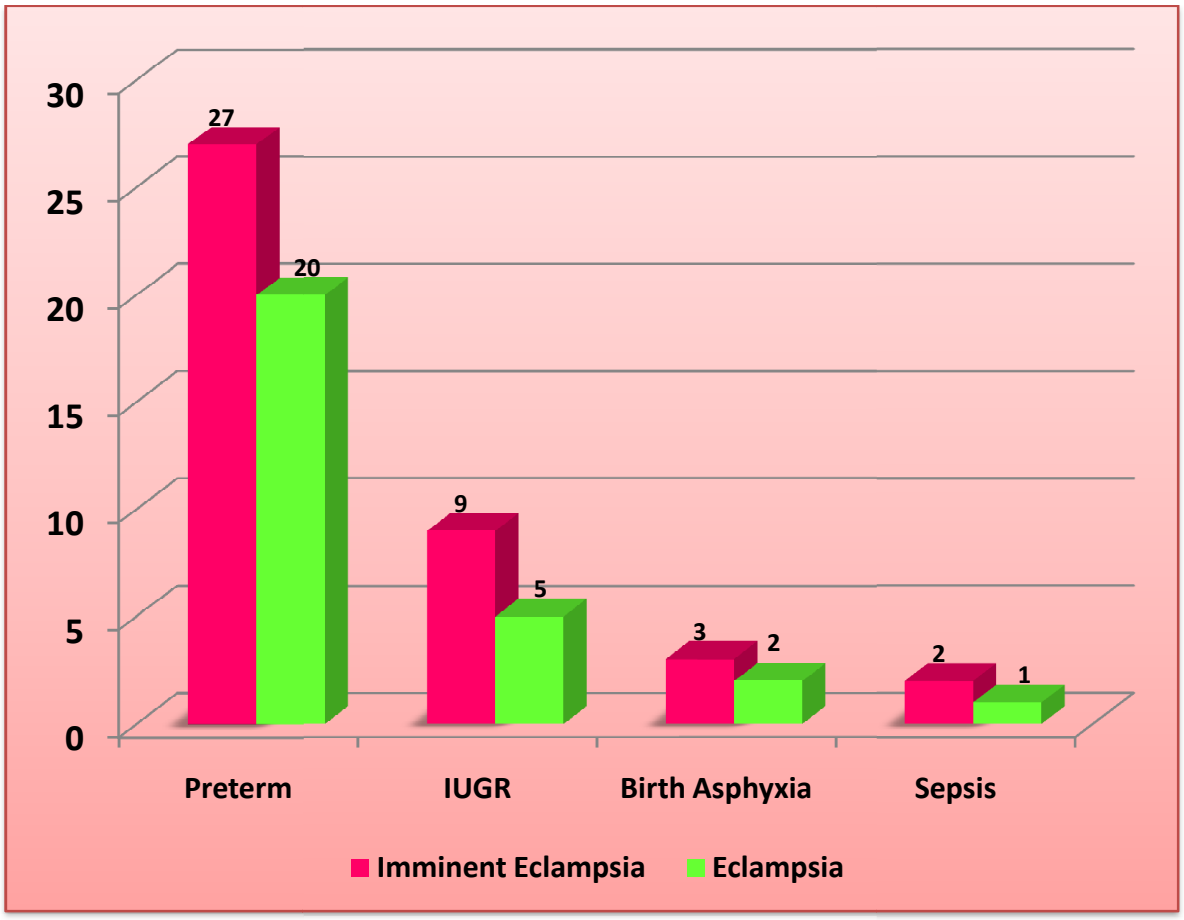
NEONATAL OUTCOME

Complication	Imminent Eclampsia	%	Eclampsia	%
Alive	41	74.5	42	80.76
Spont. Expulsion	9	16.3	5	9.61
Dead born	5	9.09	5	9.61

* there are 2 twins in imminent eclampsia and 3 twins in eclampsia

- 41 out of 55 babies Imminent eclampsia and 42 out of 52 babies of eclampsia patient were born alive.

PERINATAL COMPLICATION



PERINATAL COMPLICATION

Complication	Imminent Eclampsia	%	Eclampsia	%
Preterm	27	49.09	20	38.46
IUGR	9	16.36	5	9.61
Birth Asphyxia	3	5.45	2	3.84
Sepsis	2	3.63	1	1.92

- 49.09% and 38.46 % of Imminent eclampsia and eclampsia had preterm babies.
- 16.36% and 9.61% of Imminent eclampsia and eclampsia had IUGR babies.
- 5.45% and 3.84% babies of Imminent eclampsia and eclampsia group had Birth Ashpyxia.
- 3.63% and 1.92% babies of Imminent eclampsia and eclampsia group had Sepsis.

PERINATAL MORTALITY

Dead born >28wks		Dead with 7 days		Total			
Imminent	Eclampsia	Imminent	Eclampsia	Imminent %		Eclampsia %	
5	5	8	6	13	23.6	11	21.15

- Perinatal death in imminent eclampsia and eclampsia were 23.6% and 21.15%.

DISCUSSION

DISCUSSION

Incidence

Pradeep[28] M.R et al. 2013, in their study states that in developing countries incidence of eclampsia varies between 1 in 100 to 1 in 1700 pregnancies. **Anuja Bhalerao[29] et al. 2013,** in their study states that Incidence of Eclampsia in India 0.94 to 1.8% and Incidence in their study was 0.9%.

Incidence of Eclampsia in our study was 0.90% which was comparable to our national incidence.

Type of eclampsia

Manjusha[30]. S et al. 2013, in their study had 86.95% of Antepartum eclampsia, 4.34% of Intrapartum eclampsia, 8.69% of Postpartum eclampsia. **Knight[31]. M et al 2007,** had 45% of Antepartum eclampsia, 19% of Intrapartum eclampsia, 35% of Postpartum eclampsia in their study.

Antepartum eclampsia in our study is 65.30% comparable to above study indicating Antepartum eclampsia is more common than Intrapartum and postpartum eclampsia.

Age

Pradeep[28] M.R et al 2013 Eclampsia is more prevalent at the age 19-24yrs. **Manjusha[30]. S et al 2013** Eclampsia is common in age group of 21-25yrs.

41.5% of Imminent eclampsia and 63.3% of eclampsia patient were in age group of 20 – 25 years in our study. Age group in our study was comparable to the above studies. Eclampsia is common in young age group**.**

Socio – economic status

Pradeep[28] M.R et al. 2013, in their study states that eclampsia is common in low socio economic status.

In our study imminent eclampsia and eclampsia were common in class V, 79.25% and 65.30% respectively.

Booking Status

In the study of **Sunita[32] T.H et al. 2013,** and **Pradeep[28] M.R et al. 2013**, 95% and 88% of eclamptic cases were unbooked respectively.

In our study 73.46% of eclampsia group were unbooked and 56.6% of imminent eclampsia were unbooked. Imminent eclampsia and eclampsia were common among patient who didn't have proper antenatal care. By proper antenatal care the incidence of eclampsia can be reduced.

Parity

In the study of **Manjusha[30]. S et al. 2013,** 56% of eclamptic patient were primigravida and 43.3% were multigravida and in the study of **Sunita[32] T.H et al. 2013,** 79% of elcamptic patient were Primigravida and 21% were multigravida.

In our study 56.6% cases of imminent eclampsia and 53.06% cases of eclampsia were primi, comparable to above studies. Imminent eclampsia and eclampsia were common in primigravida.

Gestational Age

In the study done by **Pradeep[28] M.R et al. 2013,** 94% eclampsia was occurred in IIIrd trimester of which 42% in term patients.

In our study 48.97% of eclamptic patient were above 37wks of gestational age. Eclampsia is common in third trimester.

Prodromal symptoms

In study of **Stefan[21] C. Kane et al 2013.** 56% of patient who developed eclamptic seizures had headache and 15% of patients were affected by Cortical blindness. **James[33].J.Walker et al 2000,** on their study had 80% of women with prodromal symptoms before seizure.

In the study of **Dar[34] es Salaam et al 2010**, 90% of eclamptic women have prodromal symptoms of which patients with visual disturbance developed seizures within 12 hrs and for other prodromal symptoms, seizures occurred even upto 7 days.

In our study headache was common prodromal symptom in about 90.57% of imminent eclampsia patient and 89.79% of eclampsia patient. 15.09% of imminent eclampsia patient and 16.3% of eclampsia patient had

blurring of vision. By prompt identification and management at this imminent state greater number of eclampsia can be prevented.

BP at the time of admission

In the study conducted on eclamptic patient by **Pradeep[28] M.R et al. 2013,** majority of patient had diastolic BP more than 110mmHg and in the study of **Sunita[32] T.H et al,** 68% of eclampsia patient had BP >160/110mmHg.

In our study 54.7% of imminent eclampsia and 44.9% of eclamptic patient had diastolic BP more than 110. In our study 58.96% of imminent eclampsia and 57.14% of eclampsia patient ha systolic BP >160mmHg.

The study conducted by **Matter[32] F et al,** 16% of patient had no hypertension and **Manjusha[30] S et al. 2013,** in their study states that fits can occur without preceding hypertension and or proteinuria. In the study of **James[33] J.Walker et al 2000,** 20% of patient had convulsion unexpectedly, with normal blood pressure and without proteinuria. The above study had the atypical presentation of eclampsia.

In our study four patients had atypical presentation. 1 out of 53 patient in imminent group had BP 130/80mmHg. 3 out of 49 patient of eclampsia had BP <140/90mmHg.

Urine Albumin

In the study of **Manjusha[30] S et al. 2013,** 47.82% eclamptic patient had 4+ albuminuria.

Pradeep[28] M.R et al. 2013, stzudied eclamptic patient and in his study small proportion of group had no proteinuria.

In our study 1.88% of imminent eclampsia had no albuminuria and 6.12% of eclampsia had no albuminuria. 50.94% of imminent eclampsia patient had 3+ albuminuria and 34.7% of eclampsia patient had 4+ albuminuria. Most of eclamptic patient have 3+ and 4+ albuminuria however eclampsia can occur atypically without albuminuria.

Atypical presentation in our study were 6 patient. Out of 53 cases in imminent eclampsia two cases had atypical presentation. One of the atypical case had normal blood pressure with proteinuria and other case had hypertension without proteinuria. Out of 49 cases of eclampsia. Four cases had atypical presentation. Two cases presented with Bp<140/90mmHg and no proteinuria. One cases presented with Bp<140/90mmHg with proteinuria. One cases presented with Bp 150/90mmHg without proteinuria.

Referral Details:

In our study out of 49 eclampsia cases, 42 are referred cases. Among them 41 cases received loading $MgSo_4$ in the first referring unit. The one unreceived referred case got $MgSo_4$ in our hospital. 36 patients who received $MgSo_4$ in referring unit had no further fits. Two patients had fit while on transport to our hospital, 4 patients had fit in our hospital of which one patient had status epilepticus. Giving loading dose of $MgSo_4$ (Anticonvulsant

measure) in 1st referring unit reduced further recurrence of fit and thereby reducing maternal complication.

No. of convulsion

Eclampsia patient in the study of **Rajasri[35] G. et al. 2011,** 80% of patient had <5 convulsion and **Manjusha[30] S et al. 2013,** had 33.33% patient with more than 3 convulsions. In the study of **Sunita[32] T.H et al. 2013**, 38% of the patients had 1 to 2 convulsions, 49% had 3 to 5 convulsions, 12% had more than 5, 1% had coma.

In our study 38.78% of eclampsia patient had 1 convulsion, 53.06% had 2 – 5 convulsions, 6.1 % had 6 – 10 convulsions, 2.04% had >10 convulsions. No. of convulsion in our study is comparable to above study. Most of the patient of eclampsia had 2-5 convulsions and 1 patients had status epilepticus.

Admission delivery interval:

In our study average admission delivery interval was 6.15hours for imminent ecalmpsia and 4.50hours for eclampsia patient.

In our study one eclamptic patient with PRES syndrome brought in moribund state. Hysterotomy was done within 4 hours. Postoperatively she was in ventilator for one day and recovered. She regained her vision in 18hours.

In our study for another cases of eclampsia with PRES syndrome delivered by LSCS within 3hours. She recovered her vision within 12hours. A case of cerebral infarct delivered at 2.30hrs had good prognosis. A case of imminent eclampsia with pulmonary edema, treated and she delivered at 4hrs 30min. She doesn't had further complications. A case of imminent eclampsia presented with uncontrolled epistaxis delivered by caesarian section by 2hrs. She didn't had further episode of epistaxis after delivery. Early termination of pregnancy reduces the maternal morbidity.

Convulsion delivery interval

In our study eclampsia patient who delivered within 6hrs had 2.5% of perinatal death and no adverse maternal outcome. Eclampsia patient who delivered in 7 - 12hrs of first convulsion had 7.5% of perinatal death and no adverse maternal outcome. Eclampsia patient who delivered in 13 – 24hrs of first convulsion had 10.0% of perinatal death and 5.4% of maternal death. One maternal death of antepartum eclampsia patient is due to cerebral hemorrhage. Another maternal death is due to HELLP syndrome. Our study was comparable to the following study.

1)	Rajasri[35] G et al 2013	Patient who delivered within 6hrs of convulsion had least number of perinatal deaths.
2)	Sunita[32] T.H et al 2013	Patient who delivered within 6hrs of convulsion had no maternal death and least perinatal death. Patient who delivered >24hrs of convulsion had greater number of maternal and perinatal death.
3)	Thoman[36] and Colleagues (2004)	In severe pre-eclampsia and eclampsia termination of pregnancy within 12hours of admission prevents serious maternal morbidity. It was as high as 60% if the admission delivery interval was more than 48hours.
4)	Anuja[29] Bhalerao et al (2013)	In their study eclampsia patient delivered <6hrs had no maternal death and 1.82% of perinatal death. In eclampsia patient delivered 12 - 24hrs had 10.92% perinatal death and 1.82% maternal death. In eclampsia patient delivered >24hrs had 16.38% perinatal death and 3.64% maternal death.

The onset of convulsion to delivery interval was very important to decide maternal and fetal outcome

Mode of delivery

Manjusha[30] S et al. 2013, in their study had 56.25% of caesarean delivery. In the study of **Sunita[32] T.H et al. 2013**, 45% of eclamptic patient delivered by cesarean section. Eclampsia is not an indication for cesarean section but judicious and timely decision for selection of mode of delivery improve the maternal and fetal outcome.

In our study 43.4% of imminent elcampsia patient delivered by caesarian section and 37.73% of imminent elcampsia patient delivered by labour natural. In our study 40.3% of elcampsia patient was induced with PGE2. 30.61% of elcampsia patient delivered vaginally. Hysterotomy done for one patient. 59.18% of eclampsia patient delivered by caesarian section. The caesarian section is done for maternal and fetal indication and when induction failed. Caesarian rate is comparable to the above study.

Maternal complication

In the study of **Singh[24] S. et al. 2010,** 35% of the eclamptic women have serious maternal complication. Major maternal complication includes placenta abruption – 10%, Neurological deficit – 7%, pulmonary edema – 5%, cardiopulmonary arrest – 4% acute renal failure – 4% and 1 % maternal death.

The complication in our study were abruption, HELLP syndrome, ARF, status epileptics, DIC, PRES syndrome, cerebral hemorrhage, epistaxis,

cerebral infarct, PPH and death. In our study 5 patient had HELLP syndrome in which 1 patient died. 1 patient with cerebral hemorrhage died.

Imminent eclampsia and eclampsia are associated with serious maternal complications and maternal mortality.

Maternal mortality:

In our study three (6.12%) eclamptic patient and one (1.88%) of imminent eclamptic patient died.

One case of imminent eclampsia postoperatively developed pulmonary embolism and died.

A case of antepartum eclampsia had cerebral haemorrhage and died.

A case of antepartum eclampsia with HELLP syndrome had hepatic rupture and died.

A case of postpartum eclampsia had cerebral encephalopathy and died.

In study by **Anuja[29] Bhalero et al. 2013,** maternal death were 5.45% and in study by **Marina[37] Khanum et al 2004,** maternal death were 2%.

Birth Weight:

Manjusha[30] S et al. 2013, and **Rajasri[35] G. et al. 2011,** in their study 21.7% and 22% of babies born to eclampsia patient were greater than 2.5kg respectively and 78% of babies were between 1 – 2.5kg.

In our study 14.5% of imminent eclampsia and 17.30% of eclampsia patient delivered baby with birth weight >2.5kg. 16.36% babies of Imminent

eclampsia group and 30.76% babies of eclampsia group had birth weight between 2.1 - 2.5kg. 52.72% babies of Imminent eclampsia group and 42.30% babies of eclampsia group had birth weight between 1 - 2kg. **70% patient** with imminent eclampsia and eclampsia delivered **low birth weight** babies (1 to <2.5kg).

Perinatal outcome:

In the study by **Anuja[29] Bhalerao et al. 2013,** 41.82% of babies delivered by eclamptic patient were preterm, 27.27% were IUGR.

In our study 49.09% and 38.46 % of Imminent eclampsia and eclampsia patient delivered preterm babies, 16.36% and 9.61% of Imminent eclampsia and eclampsia had IUGR babies. Prematurity is common complication in babies of eclamptic patient since termination of pregnancy is done irrespective of gestational age for definite cure.

Perinatal mortality:

In the study by **Anuja[29] Bhalerao et al. 2013,** perinatal death of eclampsia group was 25.45%. **Sunita[32] T.H et al 2013,** in their study perinatal death of eclampsia group was 19%.

In our study live birth was 74.5% in the imminent eclampsia group and 80.76% in eclampsia group. Dead born was 9.09% in imminent group and

9.61% in eclamptic group. Perinatal death in imminent eclampsia and eclampsia were 23.6% and 21.15% respectively. Perinatal outcome was similar to above study.

SUMMARY

SUMMARY

- Prospective study on 102 antenatal patients conducted from August 2013 to July 2014.
- 53 cases of imminent eclampsia and 49 cases of eclampsia were studied.
- Incidence of imminent eclampsia is 0.97% and that of eclampsia is 0.90%.
- More number of unbooked cases in eclampsia.
- Eclampsia is common in young and in primigravida.
- Eclampsia is common in term gestation.
- Antepartum eclampsia is more common than intrapartum and postpartum eclampsia.
- 90% of imminent eclampsia and eclampsia patient had prodromal syndrome.
- 29 out of 53 patient of imminent eclampsia and 22 out of 49 of eclamptic patient had diastolic BP >110mmHg.
- 31 out of 53 patient of imminent eclampsia and 28 out of 49 of eclampsia patient had systolic BP >160mmHg.
- 39 out of 53 patient of imminent eclampsia and 32 out of 49 of eclampsia patient had 3+ or 4+ proteinuria.
- 4 patient of eclampsia and 2 patient of imminent eclampsia had atypical presentation.

- 53.06% of patient had 2 – 5 convuslions.
- 42 out of 49 patient of eclampsia were referred our hospital and received $MgSo_4$ in first referring unit. Receiving of $MgSo_4$ earlier helps in prevention of recurrence of fits and hence reducing maternal complication.
- Average admission delivery of eclampsia group in our study is 4.50hours. Early termination of pregnancy reduce maternal and fetal morbidity and mortality.
- In our study eclampsia patient who delivered within 6hrs had 2.5% of perinatal death and no adverse maternal outcome. The onset of convulsion to delivery interval was very important to decide maternal and fetal outcome
- In our study, caesarian section is higher. Liberalization of LSCS is done in expectation of better maternal and fetal outcome. Liberalization of LSCS has been possible due to marked improvements in anesthesiology.
- 49.09% and 38.46% of babies delivered by imminent ecalmpsia and eclampsia were preterm babies.
- 70% patient with imminent eclampsia and eclampsia delivered low birth weight babies.
- 5.66% of with imminent eclampsia and 4.08% of eclampsia patient had HELLP syndrome.

- Maternal mortality in our study group was 4 patients. 2 patient of antepartum eclampsia, 1 patient of postpartum eclampsia and 1 patient of imminent eclampsia had died.
- Perinatal death in imminent eclampsia and eclampsia were 23.6% and 21.15% respectively.

CONCLUSION

CONCLUSION

By analyzing the determinants of eclampsia for better maternal and fetal outcome, the crucial factors are duration between the convulsion to first treatment and convulsion delivery interval which are achieved by early accessibility to receive magnesium sulphate in nearby referring unit and decision for early termination of pregnancy at tertiary level hospitals.

As compared to the developed countries the incidence of eclampsia is high in India. It has a great impact on maternal and fetal morbidity and mortality. Early identification and treatment of this dreadful disease at the imminent state itself can reduce the complication.

Proper antenatal care, early detection of pre-eclampsia, health education about imminent symptom to patient and prompt management of pre-eclampsia are essential steps for prevention of eclampsia.

In current status on preventive aspect of eclampsia, atypical presentation should also be considered for which new screening and diagnostic tools has to be developed.

ANNEXURES

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MASTER CHART
IMMINENT ECLAMPSIA PATIENTS

S.No.	Name	Age	IP No.	Socio -economic status	Booking Status	Referral Status	Parity	Gestational Age	Prodromal Syndrome	BP	Urine Albumin	Received $MgSo_4$ before admission	No. of Convulsion	Mode of Induction	Mode of delivery	Admission delivery interval	Convulsion Delivery Interval	Maternal Complication	Fetal Alive	Birth Weight Kg	Complications	Out come
1	Karthigaivani	19	23106	V	UB	D.A	Primi	38	+	170/90	2+	-	-		LSCS	2	-		A A	2.2 2	IUGR IUGR	Good Good
2	Mahalakshmi	18	22698	V	UB	Ref	Primi	37	+	156/104	4+	+	-		Outlet Forceps	6	-		A	3.2	-	Good
3	Baragath Nisha	31	23119	V	B.O	Ref	Primi	35	+	180/96	3+	-	-	Gel	LN	7	-		A	1.7	Preterm	Good
4	Eswari	32	22396	IV	UB	Ref	$G_2P_1L_1$	24	+	180/90	4+	-	-	Gel	Spont Exp	8	-		Spont. Expul	600 gm	-	Spont. Expul
5	Kaliyammal	27	23012	V	UB	Ref	$G_3P_1L_1A_1$	38	+	140/110	1+	+	-	Gel	LSCS	7	-		A	2	IUGR /Birth Asphyxia	Good
6	Praveena	22	26121	III	B.O	Ref	Primi	37	+	150/100	3+	-	-	Gel	LN	10	-		A	2.4		Good
7	Buela Sundari	24	26347	V	UB	Ref	Primi	32	+	158/116	2+	+	-	Gel	LN	9	-		A	1.1	Preterm	Poor
8	Abirami	20	26275	V	B	D.A	Primi	39	+	172/92	4+	-	-		LSCS	2.5	-		A	3.2	-	Good
9	Kanagalakshmi	27	26435	V	UB	Ref	$G_2P_1L_1$	24	+	170/110	Nil	-	-	Gel	Hysterotomy	10	-		Spont. Expul	660 gms	-	Spont. Expul
10	Pandiselvi	25	29705	V	UB	Ref	Primi	37	+	180/106	3+	+	-	Gel	LSCS	7	-		A	1.6	IUGR	Poor
11	Nithya	24	29749	V	B.O	D.A	$G_2P_1L_1$	29	+	176/90	3+	-	-	Gel	LN	8	-		A	1.5	Preterm	Good
12	Ganapathyammal	30	30048	IV	B.O	Ref	Primi	35	+	180/94	3+	+	-	Gel	LSCS	8	-	HELLP	A	1.5	Preterm	Good
13	Devaki	20	36113	V	UB	Ref	Primi	40	+	138/110	3+	-	-		LSCS	2	-		A	3	-	Good
14	Narmadha	24	36126	V	UB	Ref	Primi	32	+	146/110	4+	-	-		LSCS	2	-		A A	1.2 1.0	Preterm Preterm	Good Poor
15	Subhulakshmi	27	36160	V	B.O	D.A	Primi	35	+	168/100	3+	-	-		LSCS	2.5	-	Epstaxis	A	2.1	Preterm	Good
16	Sugapriya	19	36034	IV	B	IP	Primi	38	+	174/110	3+	-	-	Gel	LSCS	-	-		A	3	-	Good
17	Radhika	20	36676	V	B.O	Ref	$G_3P_2L_1$	32	+	180/120	4+	-	-		LN	2.5	-	ARF	D	1.2	-	Poor
18	Arokiya Selvi	30	36745	V	B.O	Ref	Primi	32	+	152/96	3+	-	-	Gel	LN	8	-		A	1.4	Preterm/ Sepsis	Poor
19	Annabackiyam	27	36756	V	UB	D.A	$G_2P_1L_1$	37	+	162/100	3+	-	-		LSCS	2	-		A	2.8	-	Good
20	Pothumani	27	36785	V	UB	Ref	$G_2P_1L_1$	24	+	166/114	1+	+	-	Gel	Spont Exp	4	-	Abruption	Spont. Expul	650 gm	-	Spont. Expul
21	Raghuvani	27	40960	V	B.O	D.A	$G_3P_2L_2$	28	+	174/120	3+	-	-	Gel	LSCS	7	-		A	1.4	Preterm	Good
22	Pandiammal	19	41316	IV	UB	Ref	G_2A_1	24	+	152/114	4+	-	-	Gel	Spont Exp	11	-		Spont. Expul	950 gms	-	Spont. Expul
23	Jeyalakshmi	28	41719	V	UB	Ref	$G_3P_1L_1A_1$	35	+	150/110	3+	-	-	Gel	LN	6	-		A	1.4	Preterm	Good

24	Sheeladevi	24	41713	V	B.O	D.A	Primi	38	+	180/116	2+	-	-		LSCS	4	-		A	2.3	-	Good
25	Pandieswari	19	1463	V	B.O	Ref	Primi	37	+	158/120	3+	-	-	Gel	LN	7	-		A	1.8	IUGR	Poor
26	Hemalatha	25	1518	IV	UB	Ref	$G_2P_1L_1$	34	+	172/120	4+	-	-	Gel	LN	7	-	DIC	D	1.4	Preterm	Poor
27	Lingaeswari	29	1678	V	B	D.A	Primi	39	+	130/80	3+	-	-		LSCS	2	-	-	A	3.1	-	Good
28	Nagalakshmi	28	4005	V	UB	Ref	G_4A_3	32	+	168/98	3+	-	-	Gel	LN	9	-		A	1.4	IUGR/Preterm	Good
29	Veliyammal	27	4069	V	UB	Ref	$G_2P_1L_1$	34	+	152/104	3+	-	-		LSCS	3.5	-		A	1.8	Preterm	Good
30	Bhuvana	21	4241	III	B.O	IP	Primi	37	+	172/110	2+	-	-	Gel	LN	-	-		A	2.2	Preterm	Good
31	Rajalakshmi	28	4397	V	UB	Ref	$G_2P_1L_1$	36	+	180/106	3+	-	-		LSCS	2	-		A	1.2	Preterm/ Sepsis	Poor
32	Amarjothi	23	7095	V	UB	Ref	$G_2P_1L_1$	29	+	190/110	4+	+	-	ARM + Synto	LN	5	-	Abruption PPH	D	1.2	Preterm	Poor
33	Tamilselvi	21	7213	IV	B.O	Ref	Primi	33	+	156/100]	2+	+	-		LSCS	2	-	HELLP	A	2.2	Preterm	Good
34	Sathya	24	7491	V	UB	D.A	G_2A_1	28	+	174/104	3+	-	-		Spont Exp	8	-		Spont. Expul	950 gms	-	Spont. Expul
35	Muniyammal	31	7681	V	UB	Ref	$G_6P_5L_0$	24	+	170/120	1+	-	-	Gel	Spont Exp	12	-		Spont. Expul	560 gms	-	Spont. Expul
36	Kowsalya	30	11912	V	UB	Ref	$G_2P_1L_0$	39	+	154/100	3+	-	-		LN	5	-		A	2.8	-	Good
37	Gomathy	26	11277	V	B.O	D.A	Primi	36	+	140/110	3+	+	-	Gel	LSCS	9	-		A	1.7	Preterm	Good
38	Banu	24	11964	V	UB	Ref	Primi	28	+	170/112	3+	-	-	Synto	LN	3	-	HELLP	D	1	-	Poor
39	Sujatha	30	12183	V	B.O	Ref	Primi	38	+	152/116	4+	+	-		LSCS	3	-		A	2.6	-	Good
40	Selvi	22	12938	V	UB	D.A	Primi	30	+	180/106	1+	-	-	Gel	Spont Exp	4.3	-	Pulmonary Edema	Spont. Expul	850 gms	-	Spont. Expul
41	Maria	26	15034	V	UB	Ref	Primi	22	+	180/120	1+	+	-	Gel	Spont Exp	3	-	ARF	Spont. Expul	750 gm	-	Spont. Expul
42	Yasmintatima	19	14988	V	B.O	Ref	Primi	32	+	154/110	4+	-	-	Gel	LN	10	-		D	1.8	Preterm	Poor
43	Pusphalatha	22	15339	V	B.O	Ref	Primi	37	+	176/90	3+	+	-	Gel	LN	7	-		A	1.6	IUGR/Preterm	Good
44	Palaniammal	38	19060	IV	UB	D.A	Primi	34	+	170/110	2+	-	-		LSCS	8	-		A	1.4	Preterm	Good
45	Seethalakshmi	31	19093	V	UB	Ref	$G_3P_2L_1$	30	+	184/126	3+	-	-		LN	6	-		A	1.2	Preterm	Good
46	Jeyanthi	18	19151	IV	B.O	Ref	Primi	38	+	154/114	3+	-	-		LSCS	3	-		A	2.5	IuGR	Good
47	Kalpana	22	19882	V	UB	D.A	$G_3P_2L_1$	34	+	136/108	3+	-	-		LSCS	4	-		A	1.8	Preterm	Good
48	Priya	22	23405	V	B	IP	Primi	35	+	184/104	4+	-	-		LSCS	-	-		A	1.9	IUGR	Good
49	Nithya	18	23274	V	UB	Ref	Primi	37	+	156/106	2+	-	-	Gel	LN	7	-		A	2.1	Birth Asphyxia	Poor
50	Mary	21	23775	V	UB	Ref	$G_3P_2L_1$	26	+	186/120	4+	-	-		Spont Exp	9	-		Spont. Expul	900 gms	-	Spont. Expul
51	Durgadevi	25	23887	IV	B.O	Ref	$G_2P_1L_1$	38	+	152/110	3+	-	-		LSCS	3	-	Pulmonary Embolism and died	A	2.2	-	Good
52	Alagumani	19	27231	V	B.O	Ref	Primi	32	+	158/120	3+	-	-		LN	8	-		A	1.8	Preterm	Good
53	Menaka	22	27946	V	UB	D.A	$G_5P_2L_2A_2$	38	+	170/120	1+	-	-		LN	3	-		A	2	Birth Asphyxia	Poor

UB - UNBOOKED B – BOOKED REF - REFERRED CASE DA - DIRECT ADMISSION IP - IN PATIENT A - ALIVE

D - DEAD BORN GEL – PGE2gel SYNTO- OXYTOCIN SPONT.EXPUL - SPONTANEOUS EXPULSION

MASTER CHART
ECLAMPSIA PATIENTS

S.No.	Name	Age	IP No.	Socio -economic status	Booking Status	Referral Status	Parity	Gestational Age	Prodromal Syndrome	BP	Urine Albumin	Received $MgSo_4$ before admission	No. of Convulsion	Mode of Induction	Mode of delivery	Admission delivery Interval	Convulsion Delivery Interval in hours	Maternal Complication	Fetal			
																			Alive	Birth Weight Kg	Complications	Out come
1	Ganga	27	22516	V	UB	Ref	$G_2P_1L_1$	38	+	160/106	2+	+	4	Gel	LN	5	8	-	A	2.7	-	Good
2	Akila	26	22538	V	BO	Ref	Primi	37	+	150/90	1+	+	2	Gel	LSCS	6.5	9	-	A	2.4	-	Good
3	Sumitha	19	23308	V	UB	Ref	Primi	40	-	140/102	2+	+	1	-	LSCS	3	4	PPH	A	3	-	Good
4	Lalitha	24	26463	IV	UB	Ref	$G_2P_1L_1$	36	+	140/90	2+	+	2	-	LSCS	3	6	-	A	1.9	Preterm	Good
5	Annalakshmi	25	26525	V	BO	Ref	Primi	38	+	130/98	4+	+	1	-	LSCS	2.5	9	-	A	2.2	IUGR	Good
6	Nathiya	24	26598	V	UB	Ref	Primi	37	-	140/100	4+	+	2	-	LSCS	3	13	HELLP. DIED	D	2.8		Poor
7	Kowslya	25	30095	V	UB	Ref	Primi	38	+	170/130	3+	-	5	Gel	LSCS	2.5	9	-	A	2.9	Birth Asphyxia / MAS	Poor
8	Ambikavathy	21	29919	IV	UB	Ref	Primi	39	+	160/110	2+	+	1		LSCS	3.5	5	-	A	2.5	-	Good
9	Veeranagu	24	29660	V	UB	Ref	Primi	26	+	140/108	3+	+	3	Gel	Spont Exp	7	9	DIC	Spont. Exp	505 gms	-	Spont.Exp
10	Kanagalakshmi	27	36220	IV	B	Ref	$G_2P_1L_1$	27	+	150/110	3+	+	2	Gel	Spont Exp	6.5	9	-	Spont. Exp	560 gms	-	Spont.Exp
11	Nagajothy	22	36242	V	UB	Ref	Primi	38	+	130/70	Nil	+	2		LSCS	3.5	7		A	3.1	-	Good
12	Kalaiarasi	26	36303	V	UB	Ref	$G_2P_1L_1$	35	+	200/120	4+	+	3		LSCS	2.5	12	HELLP	D	1.5	Preterm/ IUGR	Poor
13	Maniammal	22	36607	V	BO	Ref	Primi	24	+	190/140	2+	+	3	-	Hysterotomy	4	7	PRES	Spont. Exp	500 gms	-	Spont.Exp
14	Amutha	22	36866	IV	UB	Ref	Primi	38	+	170/116	3+	+	1	Gel	LSCS	8	14	-	A	2.3	-	Good
15	Kavitha	20	36948	V	UB	D.A	Primi	34	-	180/100	2+	-	2		LSCS	1.5	3	-	A A	1.9 1.7	Preterm Preterm	Good Good
16	Vairamnai	22	41846	V	UB	Ref	$G_2P_1L_1$	37	+	150/118	4+	+	2		LSCS	3	4	-	A	2.1	-	Good
17	Venmalar	26	42244	III	BO	Ref	Primi	38	+	140/110	1+	+	1	Synto	LN	7	9	-	A	2.7	-	Good
18	Petichiammal	30	41894	V	UB	Ref	Primi	32	+	160/116	4+	+	1	Gel	LSCS	7	14	-	A	1.9	Preterm	Poor
19	Swetha	26	1837	III	BO	Ref	$G_2P_1L_1$	38	+	170/120	4+	+	4		LSCS	2.5	6	-	A	2.4	-	Good
20	Sasidevi	26	1739	IV	UB	Ref	$G_2P_1L_1$	28	+	180/120	3+	+	1	Gel	LN	8	15	-	A	1.2	Preterm/Sepsis	Poor
21	Chellam	31	2002	III	UB	Ref	$G_2P_1L_1$	32	+	140/90	2+	+	4		LSCS	3	8	PRES	A	1.5	Preterm	Good
22	Tamilsevli	22	4071	V	UB	Ref	Primi	31	+	200/108	4+	+	>10		LSCS	4	8	Status Epilepticus	A	1.5	Preterm/ Birth Asphyxia	Poor

23	Kalamani	20	4930	V	BO	D.A	$G_2P_1L_0$	38	+	170/116	3+	-	2		LSCS	2	4	-	A	2.3	IUGR	Good
24	Gajalakshmi	27	4430	V	UB	Ref	G_2A_1	37	+	160/96	3+	+	2		LSCS	8	9	ARF	A	2.3	-	Good
25	Selvi	23	7923	V	UB	Ref	$G_2P_1L_1$	32	-	140/100	3+	+	2	Gel	LN	2.5	14	Cerebral Heamorrhage death	A	1.9	Preterm	Good
26	Nagalakshmi	21	8141	V	UB	Ref	Primi	31	+	120/70	Nil	+	1		LSCS	4	7	-	A	1.2	Preterm	Good
27	Mahalakshmi	21	7992	IV	UB	D.A	Primi	37	+	190/120	3+	-	2	Gel	LN	8	9	-	A	2.4		Good
28	Vimala	21	12278	IV	BO	Ref	Primi	35	+	200/130	4+	+	9	-	LSCS	2	7	-	A A	1.8 1.6	Preterm Preterm	Good Good
29	Masilamani	20	11931	IV	UB	Ref	Primi	35	+	190/140	3+	+	3	-	LSCS	9	16	-	A	1.8	Preterm	Good
30	Sasikala	24	12859	V	UB	Ref	$G_4P_1L_1A_2$	36	+	210/130	4+	+	7	Gel	LN	7	8		A	2	Preterm	Good
31	Mythily	22	15679	V	UB	Ref	$G_4P_1L_1A_2$	36	+	150/100	3+	+	3	-	LSCS	3	8	Cerebral Infaract	A	1.7	Preterm	Poor
32	Ramuthai	24	15918	V	UB	D.A	$G_2P_1L_1$	39	-	170/120	4+	-	2	-	LN	5	7	-	A	2.5		Good
33	Vasantham	23	22685	V	UB	Ref	Primi	28	+	150/120	3+	+	3	Gel	LN		4	-	D	1.2	Preterm	Poor
34	Nagajothy	27	29830	IV	UB	IP	Primi	34	+	160/110	4+	-	1	-	LSCS	-	2	-	A A	1.8 1.7	Preterm Preterm	Good
35	Rakku	24	37074	V	B.O	Ref	Primi	26	+	150/106	3+	+	1	Gel	Spont. Expul	-	6	-	Spont. Expul	600 gms	-	Spont. Expul
36	Kavitha	30	1673	V	UB	Ref	$G_2P_1L_1$	38		156/104	2+	+	1	Synto	LSCS	-	4	-	A	2.5	-	Good
37	Ranjitha	19	8096	IV	UB	Ref	Primi	39	+	160/90	4+	+	1	Synto	LN	-	3	-	A	2.6	-	Good
38	Mahesh	31	23449	V	UB	Ref	$G_2P_1L_1$	32	+	110/70	1+	+	1	-	LN	-	-	-	D	1.8	-	Poor
39	Jeyanthi	27	27947	V	UB	Ref	$G_2P_1L_1$	36	+	150/100	3+	+	1	-	LN	-	-	-	A	2.5	Preterm	Good
40	Manimegalai	23	29730	IV	B.O	Ref	$G_2P_1L_1$	37	+	170/120	4+	+	3	-	LSCS	-	-	-	A	2.6		Good
41	Veerachinnammal	22	36352	V	UB	IP	Primi	34	+	160/102	3+	-	1	-	LSCS	-	-	-	A	1.7	Preterm	Good
42	Revathy	24	37602	III	B.O	Ref	$G_2P_1L_1$	39	-	150/102	+	+	1	-	LN	-	-	-	A	2.5	-	Good
43	Shanthamani	24	43048	V	B	Ref	$G_2P_1L_1$	38	+	170/100	2+	+	1	-	LSCS	-	-	-	A	2.8	-	Good
44	Suriya	21	2759	V	UB	Ref	Primi	38	+	200/140	4+	+	6	-	LSCS	-	-	Cerebral Encephalopathy . Died	A	2.5	Birth Asphyxia	Poor
45	Santhanam	23	5959	V	UB	Ref	$G_3P_2L_2$	37	-	160/100	3+	+	2	-	LN	-	-		A	2.6	-	Good
46	Laxmithai	21	7541	V	UB	Ref	$G_2P_1L_1$	26	+	180/130	1+	+	1	Gel	Spont. Expul	-	-	Pulmonary Edema	Spont. Expul	600 gms	-	Abortion
47	Jeyalakshmi	22	13514	IV	UB	Ref	$G_2P_1L_0$	34	+	160/106	4+	+	2	-	LN	-	-	-	D	1.3	Preterm	Poor
48	Priya	29	16499	V	B.O	Ref	Primi	38	+	150/90	Nil	+	2	-	LSCS	-	-	-	A	2.4	IUGR	Good
49	Seethalakshmi	26	21157	IV	UB	IP	Primi	36	+	160/100	2+	-	1		LN	-	-		A	1.8	IUGR	Good

UB - UNBOOKED B – BOOKED REF - REFERRED CASE DA - DIRECT ADMISSION IP - IN PATIENT A - ALIVE

D - DEAD BORN GEL – PGE2gel SYNTO- OXYTOCIN SPONT.EXPUL - SPONTANEOUS EXPULSION

Ref. No. 2443/ME1/14

Government Theni Medical College
Theni. Dated: 26.08.2014

Institutional Ethical Committee:
Convenor:

Dr. R.M. Raja Muthiah, M.S., M.Ch.,
Dean
Govt. Theni Medical College
Theni

Sub: Medical Education – Govt. Theni Medical College,
Theni – Ethical Committee – Minutes – Communicated – Reg.

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The Ethical Committee Meeting of the Govt. Theni Medical College, Theni was held at 12.00 noon on 26.08.2014 at Conference Hall, Near Dean's Chamber, Government Theni Medical College, Theni.

The following Members of the Committee have attended the Meeting.

1.	**Convener**	:	**Dr.R.M. Raja Muthiah, M.S., M.Ch., DEAN**
2.	**Member Secretary**	:	**Dr. K. Kathirkamu, M.S., Deputy Superintendent**
4.	**Members**		
	Professor of Medicine	:	**Dr. P. Purushothaman, M.D.,**
	Professor of Surgery	:	**Dr. R. Murugesan, M.S.,**
	Professor of Obs. & Gynaec.	:	**Dr. Thangamani, M.D., O.G.,**
	Professor of Micro Biology	:	**Dr. K.M. Mythreyee, M.D.,**
5.	**Chairman (Private Consultant)**	:	**Dr. Paulraj, M.D.,** Ramya Clinic, Periyakulam Road, Theni.
6.	**Lawyer**	:	**Thiru.K.Murugesan, B.Com., B.L.,** S/o.Kamaraj, Ambedkar Nagar, Varusanadu, Theni District.
7.	**Sociologist**	:	**Sr. Anaestescia** Director, Jeevan Jothi Hospital Community Care Centre, Periyakulam Road, Kailasapatti, Theni Dist.
8.	**Public**	:	**Mr. P. Deenadhayalan, M.A.,** Land Lord, Koduvilarpatti, Theni District.

The following Project was approved by the Committee:

Name of the PG	Course	Name of the Project	Remarks
Dr. K. Meena	PG in M.S. Dept. of Obs. & Gynaec. Govt. Theni Medical College, Theni	Study on Determinents and current status of Imminent Eclampsia and Eclampsia.	Approved

Please note that the investigator should adhere the following: He/she should get a detailed informed consent from the Patients/participants and maintain Confidentially.

11. He/she should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution.
12. He/she should inform the Institution Ethical Committee in case of any change of study procedure site and investigation or guide.
13. He/she should not deviate for the area of the work for which applied for Ethical Clearance. He/She should inform the Institution Ethical Committee immediately, in case of any adverse events or any serious adverse reactions.
14. He/she should abide to the rules and regulations of the institution.
15. He/she should complete the work within the specific period and apply for if any extension of time is required. He/she should apply for permission again and do the work.
16. He/she should submit the summary of the research work to the Ethical Committee on completion of the work.
17. He/she should not claim any funds from the institution while doing the work or on completion.
18. He/she should understand that the members of Institutional Ethical Committee have the right to monitor the work with prior intimation.

26/8/14

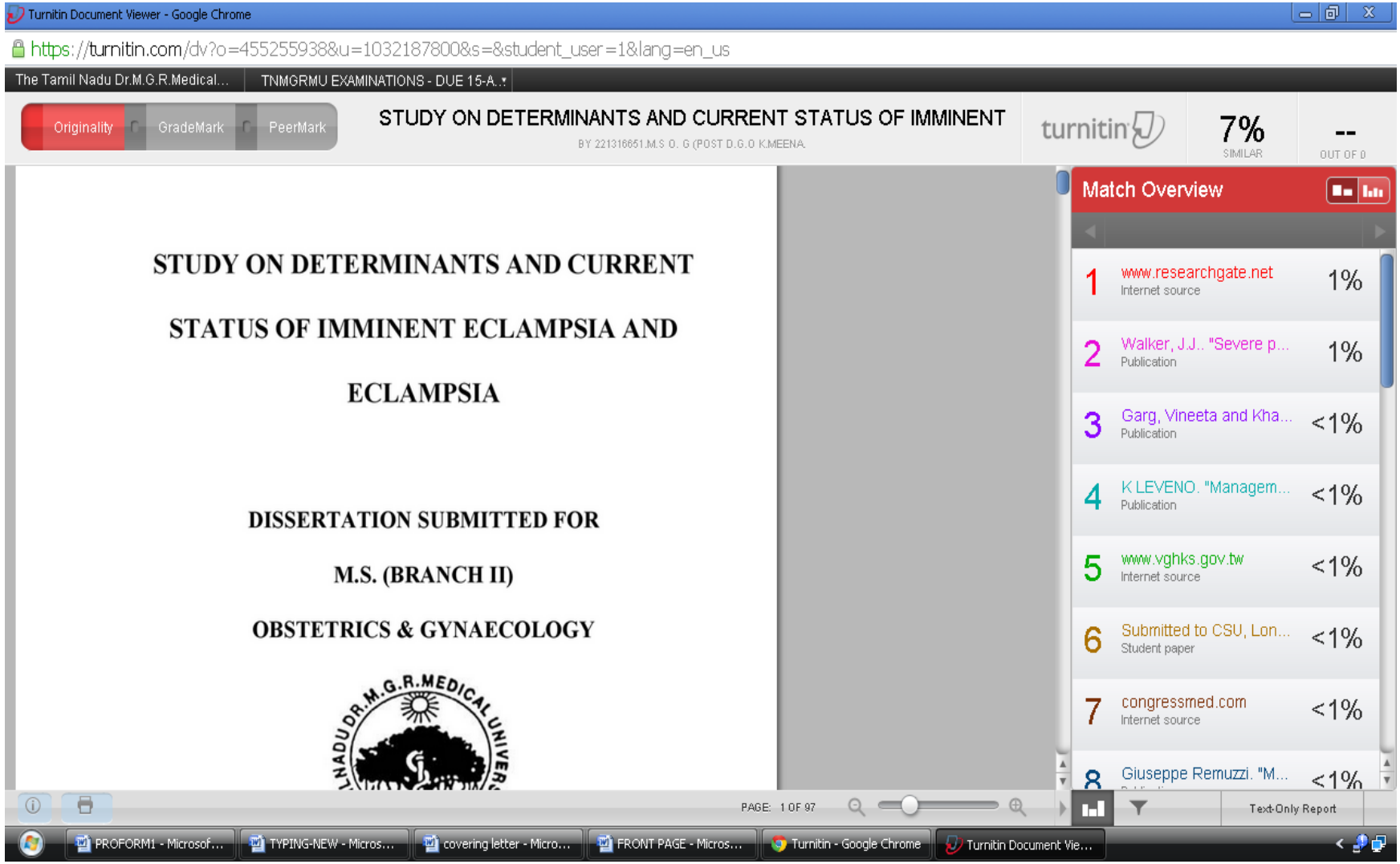
Chairman

26.8.14

Convenor

To

The above PG Student – through Head of the Department concerned.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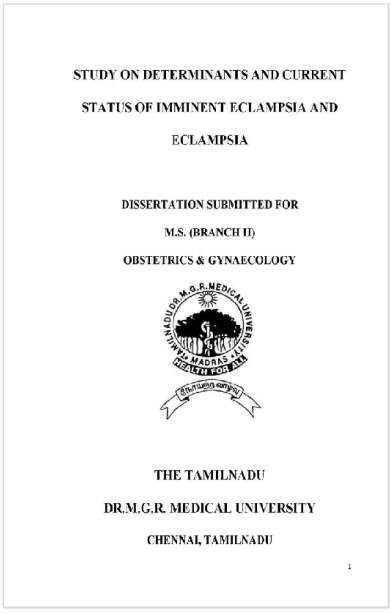

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